Bulletin of the Experiment Station of the Hawaiian Sugar Planters' Association

EXPLANATION OF FRONTISPIECE.

REACTION TO MOSAIC AS SHOWN BY THE TISSUES IN THE LEAVES OF FOUR VARIETIES OF CANE.

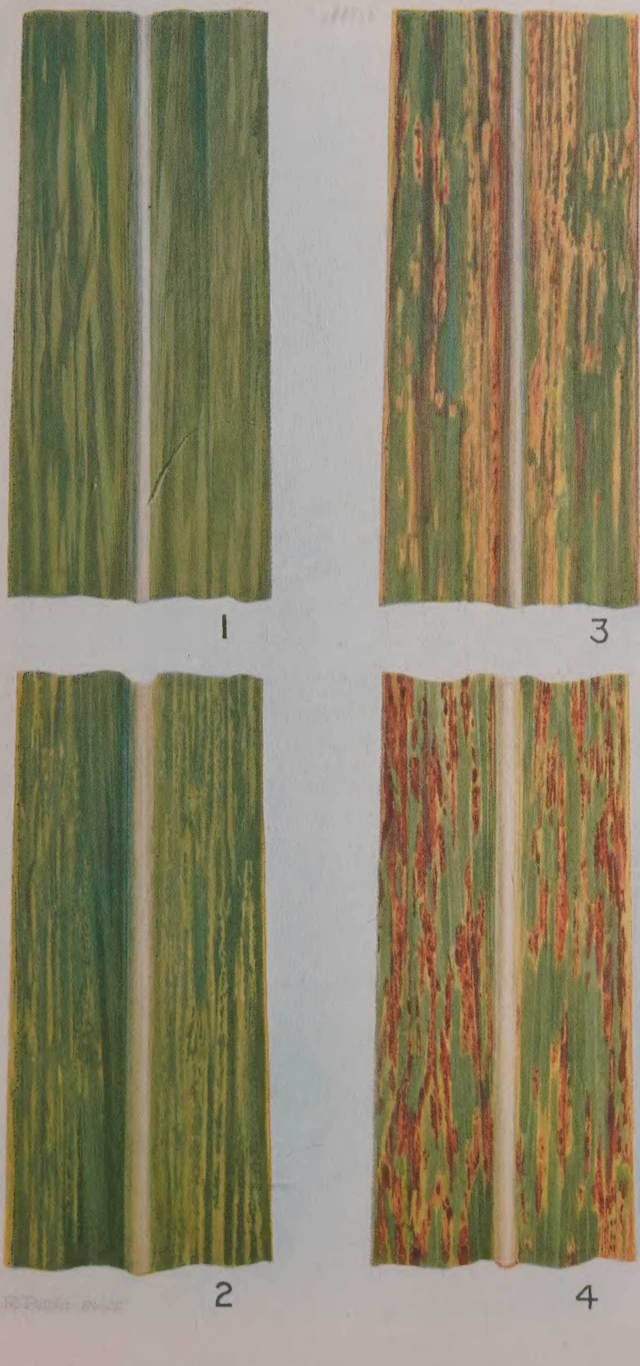
Figure 1—Striped Tip. The pattern is very large and very pronounced. The tissues in the affected areas improve as the leaf ages until in an old leaf it is sometimes difficult to distinguish the mottlings.

Figure 2—Hawaii No. 355. In this variety the tissues within the affected areas deteriorate as the leaf ages, becoming quite yellow in the mature leaf, but still remaining alive throughout the life of the leaf.

Figure 3—Hawaii No. 27. In H. 27 the tissues within the affected areas not only deteriorate, but actually die prematurely, so that the older leaves on a diseased shoot exhibit patches of dead and discolored tissue as here shown.

Figure 4—Hawaii No. 207. The affected tissues in the leaves of this variety show no ability whatsoever to recuperate after the leaf expands, but soon die, producing a striking pattern in yellow and red which makes a diseased shoot very conspicuous in the field.

FRONTISPIECE



THREE MAJOR CANE DISEASES: MOSAIC, SEREH, AND FIJI DISEASE.

By H. L. Lyon

The writer began an intensive investigation of these three serious cane diseases in the fall of 1910 and devoted practically all of his time to their study up to 1914, when he was compelled to drop the work and has at no time since been able to resume it. Extensive notes and considerable experimental data were accumulated, but the work had not reached such a stage of completion as seemed to warrant its publication. The appearance and spread of Mosaic in the southern United States and throughout the West Indies, and the invasion of the Philippine Islands by Fiji disease have brought these two diseases into prominence and created the desirability of clearly differentiating them from the all-including Sereh of Java. The following discussion is offered at this time in the hope that it may help to accomplish this purpose and otherwise be of assistance to those now conducting critical studies of these diseases. The descriptions here given are in most part but portions of manuscripts prepared prior to 1914. Little space is allotted to the discussion of possible causes, the chief aim being to supply adequate descriptions of the critical symptoms.

Sereh was the first of these diseases to be recognized and it inspired a very extensive literature wherein most of the symptoms of disease in sugar cane are credited to this one malady. As a result Sereh has, at some time or other, been reported in most of the sugar growing countries of the world. Much of the confusion regarding Sereh is, no doubt, due to the fact that the symptoms induced by the many cane diseases extant in Java have not been clearly differentiated. With a number of obscure cane diseases operating at the same time in a country, it must be very difficult to accurately segregate the symptoms of each. In Java they have Sereh, Mosaic, and Gumming disease, which is a combination well designed to breed confusion. Despite the fact that Sereh has received more critical study than any other known cane disease, it is quite safe to say that cause and effect in this disease are still very imperfectly known. In countries far removed from Java we find other well defined cane diseases producing one or more of the very symptoms which are cited in Java as peculiar to Sereh. When a cane is badly stunted by disease in Java the stunting is at once ascribed to Sereh, but in Hawaii Striped Tip cane is stunted by Mosaic exactly as the Cheribon variety is stunted by Sereh in Java, while in Fiji a similar stunting in the variety Daniel Dupont is induced by Fiji disease.

The only defensive measures which have proven effective against these three diseases are, (1), the use of resistant varieties of cane and, (2), the selection of healthy canes only for purposes of propagation. These defensive measures are efficacious in exact proportion to the ability of those directing them to distinguish these diseases in all of their various phases. An accurate knowledge of their symptoms is therefore the most essential equipment for defensive operations.

MOSAIC.

The sugar cane malady which is now becoming familiarly known as Mosaic has been the cause of much discussion during the past three years among those interested in the culture of sugar cane in the United States and the West Indies. The subject was introduced by Stevenson,[1] who announced the discovery of a *new* and alarming cane disease in Porto Rico. As soon as Stevenson's paper came to hand the writer prepared and forwarded to "Science" a brief criticism as follows:

"An Epiphytotic of Cane."

> In a recent number of Phytopathology (December, 1917) Stevenson describes a "new and alarming cane disease," which has appeared in Porto Rico.
>
> From his description and illustrations it is readily seen that he is dealing with a malady, concerning which there already exists a considerable literature. The disease has been known for many years in Java under the name "Gele Strepenziekte." It was first mentioned in print by van Musschenbroek [2] in 1892. In April, 1893, Wakker [3] published further notes on the disease and in July of the same year Hein [4] contributed a short article on the subject, which was illustrated by a colored plate. Since that time it has received frequent mention in the literature on the sugar cane emanating from Java. It was fully described by Wakker and Went (1898) in their book, "De Ziekten van het Suikerriet op Java," and briefly by Krüger (1899) in his book, "Das Zuckerrohr und seine Kultur." The most recent, as well as the most thorough treatment of the disease in Java has been contributed by Miss Wilbrink and F. Ledeboer [5] (1910). Their careful description and excellent colored plates should make it an easy matter for anyone to correctly diagnose the disease.
>
> Gele Strepenziekte was first noted in Hawaii in 1908. Here it is known as "Yellow Stripe disease," this being a literal translation of the Dutch name. It is an infectious chlorosis akin to the Mosaic disease of tobacco. The causal agent operates at the growing point of the stem and in the unexpanded leaves which are rolled up in the spindle. Every lateral bud is infected as it is formed, so the disease is certain to be transmitted through cuttings from affected sticks. All varieties of cane grown in Hawaii are susceptible to this disease, but some much more so than others. Likewise some varieties are far more sensitive to the disease than others; a few being rendered quite worthless when attacked, while others stand up well under the disease.
>
> In Java and in Hawaii the disease is held under practical control by the selection of healthy sticks only, for cuttings.
>
> We have authentic records of the occurrence of Yellow Stripe disease in Hawaii, Fiji, Australia, New Guinea, Java, the Philippines, Egypt, Cuba and Porto Rico.
>
> An infectious chlorosis of corn has become quite prevalent in the corn fields of Hawaii during recent years. Corn is more seriously in-

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[1] Phytopathology 7:418–425, 1917.

[2] Circulaire No. 42 der Soerabaijasche Vereeniging van 16 Oct. 1892 bl. 327.

[3] Archief voor de Java Suikerindustrie 1:4.

[4] Archief voor de Java Suikerindustrie 1:215.

[5] Archief voor de Java Suikerindustrie 18:465–518.

Figure 1—Mosaic in (a) Striped Tip; (b) Lahaina; (c) H 109.

jured by this disease than is cane by Yellow Stripe disease. There is some reason to believe that the cane and corn diseases are identical, but this conclusion has not as yet been fully demonstrated.

Harold L. Lyon.

Honolulu, Hawaii, December 20, 1917.

For some unknown reason the above criticism was not printed in "Science" and it did not seem necessary to send it elsewhere for publication as its object was soon accomplished through the circulation of type-written copies among plant pathologists in Washington, Cuba and Porto Rico who were concerned with the diseases of the sugar cane.

Since the recognition of the disease in Porto Rico it has been found in Louisiana, Florida, Cuba, Jamaica, Trinidad and Barbados, while information is available which indicates that it has been present in Argentina for at least twenty years and is now of quite general occurrence in the cane fields of that country.

Investigations of Mosaic are now being actively prosecuted by plant pathologists in Washington, D. C., Louisiana, Cuba, Porto Rico, Jamaica and Hawaii, and we may look for rapid progress in the elucidation of the serious problems in cane husbandry which this disease has created.

Diagnosis.

Primary Symptoms. The primary and critical symptom of this disease is the appearance of pale patches or blotches in the green tissues of the leaves. The blotches themselves are primarily of a uniform green throughout, but at the same time they are several shades lighter than the normal green tissues which surround them. They are not constant in size or shape even on the same leaf, although they often run very large in some cane varieties and very small in others. They are usually irregularly oval or oblong in outline, their longer axes lying parallel to the midrib. They are not confined between veins and consequently are not of uniform width throughout any considerable part of their length. They do not simulate stripes, therefore, and "Gele Strepenziekte" or "Yellow Stripe disease" is obviously an unfortunate choice of names for the malady, especially since there are other cane diseases which do cause the formation of very definite stripes in the leaves. The terms marbled, blotched and mottled describe the real appearance of diseased leaves much better than "Yellow Stripe," as can be readily seen by referring to the Frontispiece, Plate 1, and text figures 1 and 2. When our studies of this disease in Hawaii showed it to be an infectious chlorosis of the same general nature as the well-known Mosaic diseases of tobacco, tomato and other plants we adopted the more appropriate name "Mosaic," which in itself constitutes an intelligible description of the malady.

In all cane varieties the characteristic light patches on diseased leaves are very distinct when the leaves first unroll from the spindle. They are by no means equally distinct on the leaves of all varieties, but they are easily discernable in all varieties and, at this time, conform most nearly to a color type. The newly

Figure 2—Mosaic in (d) Yellow Caledonia; (e) D 1135; (f) Badila.

opened leaves are, therefore, the ones to examine when seeking symptoms of the disease.

The green tissues of the leaf-sheath are affected in the same manner as those of the leaf-blade but the blotches are always less conspicuous.

The light patches are marked off in the tissues of the leaves for some time before they unroll from the spindle; in fact the outlines of the patches are discernible as soon as the young leaves begin to turn green inside the spindle. After a leaf has unrolled the patches do not change materially in size or shape, but they usually do change considerably in color; in some varieties becoming more distinct, and in others less distinct, as the leaf grows older. As a rule this change in color consists of an intensification or a paling of the green within the blotch, but in a few varieties (H 27, H 207 and H 296), the green regularly gives place to yellow, red and brown as the leaves grow older. (See Frontispiece.) The appearance of these striking colors, "autumn tints," is due to the rapid deterioration and death of the chlorophyllous tissue in the blotches.

The blotches on diseased leaves of all ages may be more easily detected if the leaves are examined in the shade rather than in direct and glaring sunlight. Examination by transmitted light is also an aid to diagnosis, but should be supplementary to examination by reflected light for, employed alone, it sometimes leads to error.

The first indication of Mosaic displayed by a newly affected shoot is an irregular blotch or two on the last leaf to unroll from the spindle. Succeeding leaves show these blotches in increasing numbers and varying sizes. Leaves may soon appear on which the blotches cover the greater part of the leaf, running together in an irregular mosaic. In the majority of cane varieties the blotches are far less distinct on the older leaves than on the younger leaves only recently unrolled from the spindle.

The appearance of light blotches on the leaves is the primary and critical symptom of Mosaic and the only one on which a diagnosis should be based.

Secondary Symptoms. Some varieties of cane may carry Mosaic year after year without showing any symptoms except the mottling of the leaves, while other varieties may display very decided secondary symptoms. Certain of these secondary symptoms are manifested in varying degrees by several varieties, while others are displayed by only a few, or even a single variety. It is evident, therefore, that while these secondary symptoms, when present, may clearly indicate the nature of the disease they are not the ones to look for when striving to identify the disease in the field.

Mottling or marbling of the stem is the one secondary symptom most frequently displayed by diseased canes. The outer tissues of the stem, commonly termed the rind, contain a considerable amount of chlorophyll and one might reasonably expect that the mottled effect which is always produced in the green tissue of the leaf might in some cases be extended to this green tissue of the rind, and this is what actually occurs. In some varieties of cane no mottling of the rind can at any time be distinguished; in other varieties it is very indistinct and transient, disappearing entirely shortly after the internode is exposed to the light: in still other varieties it becomes apparent only after

the internode is exposed to strong light. In Lahaina cane, for instance, the mottling can hardly be distinguished at any time on diseased sticks which are protected from strong sunlight by trash and foliage, but on sticks that are well exposed the mottling often becomes very pronounced, being accentuated by the development of strong red and purplish tints in the blotches, as shown in Plate 2. While a similar production of unusual colors in the rind tissue is induced in many green canes by Mosaic, in canes which normally have red or purple rind tissue the mottling, when present, is manifested by the diminution or loss of the normal red or purple color and the blotches appear green on a dark background.

The cankering of the stem as described by the plant pathologists of Porto Rico is but the extension of this mottling of the rind to a point which results

Figure 3—Mosaic in Striped Tip. Two cuttings from diseased canes were planted alternately in the same row with two cuttings from healthy canes. The photograph shows the resulting stools ten months later. Those from diseased cuttings are very much stunted. Many of the shoots from the healthy cuttings became diseased, and in consequence did not reach normal size.

in the death of the affected tissue. No case has ever been met with in Hawaii where the injury of the rind within the blotches has been pushed to a point which resulted in the complete destruction of the tissue as it does in Porto Rico. The writer was able to study the inception of these cankers to good advantage, however, while visiting Jamaica in 1920. On a seedling variety (Barbados 6450) growing at the Sugar Experiment Station in Kingston the mottling of the rind was very pronounced and quite conspicuous on the young internodes only a short distance back from the growing point. The affected tissues died while the internode was still small and soft, forming sickly white patches in the rind. As the tissues in these patches were dead they did not grow to keep pace with the growth and expansion of the tissues within and so were soon burst asunder. The cracks or cankers thus formed in the rind afforded easy entrance to various fungi, which soon penetrated the soft tissues within the stem and brought about their speedy destruction. In this variety we see the same se-

Figure 4—Mosaic in Yellow Caledonia. Four diseased canes are shown along side of sections of equal length taken from healthy canes of the same age. Practically the entire stick is included in the case of the diseased canes, which were only one-third as long as the healthy canes.

quence of events occurring in the case of the rind tissue that we describe above for the leaf tissues in the Hawaiian seedlings numbers 207 and 296. One can readily see that canes which are cankered in this manner by Mosaic are certain to be rendered worthless. We hold ourselves extremely fortunate in that Mosaic has never produced this extreme injury in any of our cane varieties in Hawaii.

The production of small and deformed sticks, or no sticks at all, is a symptom induced in a few cane varieties by Mosaic. Among our standard varieties this symptom is common to Striped Tip and Yellow Caledonia. Mosaic causes Striped Tip to make a growth which exactly simulates the stunted growth induced in Cheribon cane by Sereh, as described on page 23, and when in 1908 we first met with a case of Mosaic in Striped Tip we were inclined to diagnose it Sereh. The occurrence in the diseased sticks of a thick red gum in many

Figure 5—Mosaic in Demerara 115. Sections from a diseased stick, showing internal masses of discolored tissue.

of their vascular bundles was an additional symptom supporting the same diagnosis. In our later experience with Mosaic in Striped Tip, however, we found that a stunted growth, such as shown in text figure 3, was characteristic of the disease in this variety. In Yellow Caledonia the stunting is not so pronounced as in Striped Tip, but diseased sticks are always more slender and much shorter than the healthy sticks and in addition they usually show longitudinal grooves or folds in the rind, as may be seen in the stick illustrated in Plate 3 and less distinctly in the sticks shown in text figure 4. These grooves in the rind of Yellow Caledonia are the nearest approach displayed by any of our cane varieties to the cankered condition induced in canes in the West Indies by Mosaic.

As a rule Mosaic does not by itself cause any marked changes in the in-

ternal tissues of the stem. In sticks long affected with the disease, however, there may sometimes be found irregular masses of internal tissue which are more or less stained and discolored; ranging from light red to a very dark brown or almost black. In no cane variety has this discolored tissue been found in all diseased sticks, but it occurs much more frequently in some than in others. The seedling cane Demerara 115 displays this symptom more frequently and more extensively than any other cane variety in our cultures, but the symptom is not uncommon in diseased canes of Striped Tip and Yellow Caledonia. In the latter variety the discolored tissue most frequently occurs directly beneath the grooves in the rind previously mentioned.

Mosaic does not, as a rule, induce an appreciable change in the size or shape of the leaves produced by a cane, but stunted shoots of Striped Tip sometimes terminate their efforts to grow by throwing a few twisted and distorted leaves in the manner characteristic of canes about to succumb to Fiji disease. (See page 37.)

Resistance and Susceptibility.

No cane variety grown for sugar production in Hawaii is immune to Mosaic. Some varieties are so very resistant to the disease, however, that for practical purposes they may be considered immune. Striped Mexican, Badila and Demerara 1135 are the best canes in this class. Yellow Caledonia is only moderately resistant, while Lahaina, Striped Tip and Demerara 117 are very susceptible to the malady. Seedling canes are almost invariably as susceptible as are their parents. This is true of H 109, which is a seedling of Lahaina. The so-called native canes, which were cultivated by the natives prior to the coming of the white man, are all very susceptible to the disease. The Striped Tip and Yellow Bamboo of the present day are in reality native canes under assumed names. The "Kavangire" cane, which is being exploited in the West Indies as an immune variety, is known as "Uba" and "Japanese cane" in Hawaii. Here it is grown for forage purposes only, being considered a cane that cannot be grown profitably for sugar production under local conditions. The true Cavengerie is a strong-growing cane, producing large, dark red sticks with distinct black stripes on many of their internodes, and dark green leaves which frequently contain stripes of albino tissue. It is one of the old standard canes of the sugar world. It is very susceptible to Mosaic.

Infection Experiments.

Despite much careful study the writer and his associates were not able to locate, by microscopic examination or cultural methods, any organism in canes suffering with Mosaic which might be responsible for the disease. Numerous experiments were conducted, however, in attempts to convey the disease by artificial means from diseased to healthy plants. The media most employed were juices extracted by pressure from various parts of affected canes, but more particularly from tissues adjacent to the growing point of the stem. Inoculation of healthy plants was attempted by applying these juices externally to all

parts, and introducing them internally at various points in the stem, eyes and spindle with a needle-syringe.

In no case did the disease appear on a high percentage of treated canes or canes from treated cuttings, and in every experiment it appeared on such a number of shoots in the checks as to invalidate the evidence of artificial infection.

Most of these experiments were conducted in the open under field conditions and in a restricted area where the disease was at all times present in nearby canes. In the few experiments where canes grown in tubs and carefully isolated in a glass house were employed, only negative results were obtained.

In all of our infection experiments we used Lahaina cane, which is, as previously noted, very susceptible to Mosaic.

Field Studies.

When we began our studies of Mosaic in sugar cane and attempted to institute measures for its control and elimination in Hawaii we met with some opposition on the ground that the disease was not infectious, but should be explained as but the manifestation of an inherent sporting tendency in a hybrid race of plants. According to this interpretation each new mottled shoot must be considered nothing more than a bud-sport. Van der Stok[1] propounded this theory in 1907, and his position was more fully explained and illustrated by Miss Wilbrink and F. Ledeboer.[2] Since the mottled canes came true from cuttings exactly as did the many well known bud-sports of the horticulturist, and in view of the fact that we were unable to isolate a causative agent or to transmit the malady at will from diseased to healthy plants, it proved a rather difficult matter to demonstrate the fallacy of Van der Stok's theory. Another hypothesis that was more easily disproved was the contention that the mottling of the leaves was due to peculiarities of the particular soil on which the cane was growing. As the infectious nature of Mosaic diseases is now generally recognized by plant pathologists we shall not attempt to marshal evidence to disprove these theories. A few miscellaneous experiments and observations are here cited, however, to show the nature and eccentric habits of the malady.

Cuttings taken from healthy canes in fields where the disease did not occur were planted in the vicinity of diseased canes. The young shoots springing from these healthy cuttings were invariably healthy at first, but as they developed, a varying percentage of them always contracted the disease. This experiment was repeated many times at our experiment grounds in Honolulu and always gave the same result except for the speed of infection and the percentage of the plants infected, in which respects they varied considerably. This was true not only when different varieties were employed, but in succeeding experiments where the same variety was used. One of the earlier experiments of this nature is illustrated and described on page 7, text figure 3.

In 1911 Mosaic did not occur on any of the standard canes on the island

[1] Archief voor de Java-Suikerindustrie, 15:581-601.
[2] Archief voor de Java-Suikerindustrie, 18:465-518.

of Kauai. On several plantations small blocks were planted with cuttings of seedling canes obtained on Oahu. These cuttings were evidently taken from diseased stools, for a large percentage of the resulting shoots showed mottling on the first leaves which they developed. In 1912 Mosaic appeared on two plantations in the Lahaina cane adjacent to the blocks of seedlings and since that time it has spread considerably on one of these plantations despite serious efforts to eliminate it.

At an early stage in our study of Mosaic it became evident that there were factors outside of the cane itself and the causative agent which determined the rate of infection. In the Hilo district of Hawaii, for instance, Mosaic, although continuously present in fields of Striped Tip, has not spread to an appreciable extent during the past ten years, while in the nearby Hamakua district of the same island the disease has repeatedly, and in the course of a few months, spread through large areas of this same cane variety.

We have records of several instances where the disease has spread rapidly in a field during a period of three or four months and then during a succeeding period of equal or greater length has not spread at all. In connection with this feature we should state, however, that it is a matter of frequent observation that during a period of rapid spreading the disease always makes greater and more rapid headway in fields of young cane than it does in fields of old cane of the same variety in the immediate vicinity.

Mosaic is not induced in the cane plant either directly or indirectly by soil conditions. It is not transmitted through the soil and it does not hold over in the soil from one planting to the next. These contentions were demonstrated an indefinite number of times by removing diseased stools and planting in their places cuttings from healthy canes. These cuttings invariably gave rise to healthy stools which remained healthy throughout several seasons. In a large field where over 90% of the canes were diseased the stools were removed and the field planted with healthy cuttings, which gave rise to a stand of cane in which the disease has never appeared.

When the disease appears in a field not previously affected it does not, as a rule, appear on every stool over large areas, but spreads sporadically through the field. This is well illustrated in the accompanying charts, text figures 6 and 7, which show the healthy and diseased stools in three blocks of seedling canes. These seedlings were all healthy when set out in the field.

The evidence of a long series of observations and experiments seems to indicate that the causal factor responsible for Mosaic in cane operates at or near the growing-point of the stem and entirely within the spindle of unexpanded leaves. As previously noted, the actual injury to the chlorophyllous tissue is accomplished while the leaf is still rolled up in the spindle, the shape and size of the blotches being determined before the leaf unrolls. It is quite evident that after the leaf has once unrolled from the spindle its tissues are not subject to further injury from the causative agent, for in the great majority of cane varieties the tissues rapidly improve. In a few varieties, such as H 207 and H 296, it is true that the tissues within the blotches continue to deteriorate after the leaf expands, but this seems to be due to lack of power in these particular varieties to recuperate from the injury received while the leaf was still in the spindle, rather than to a continuation of the action of the causative agent.

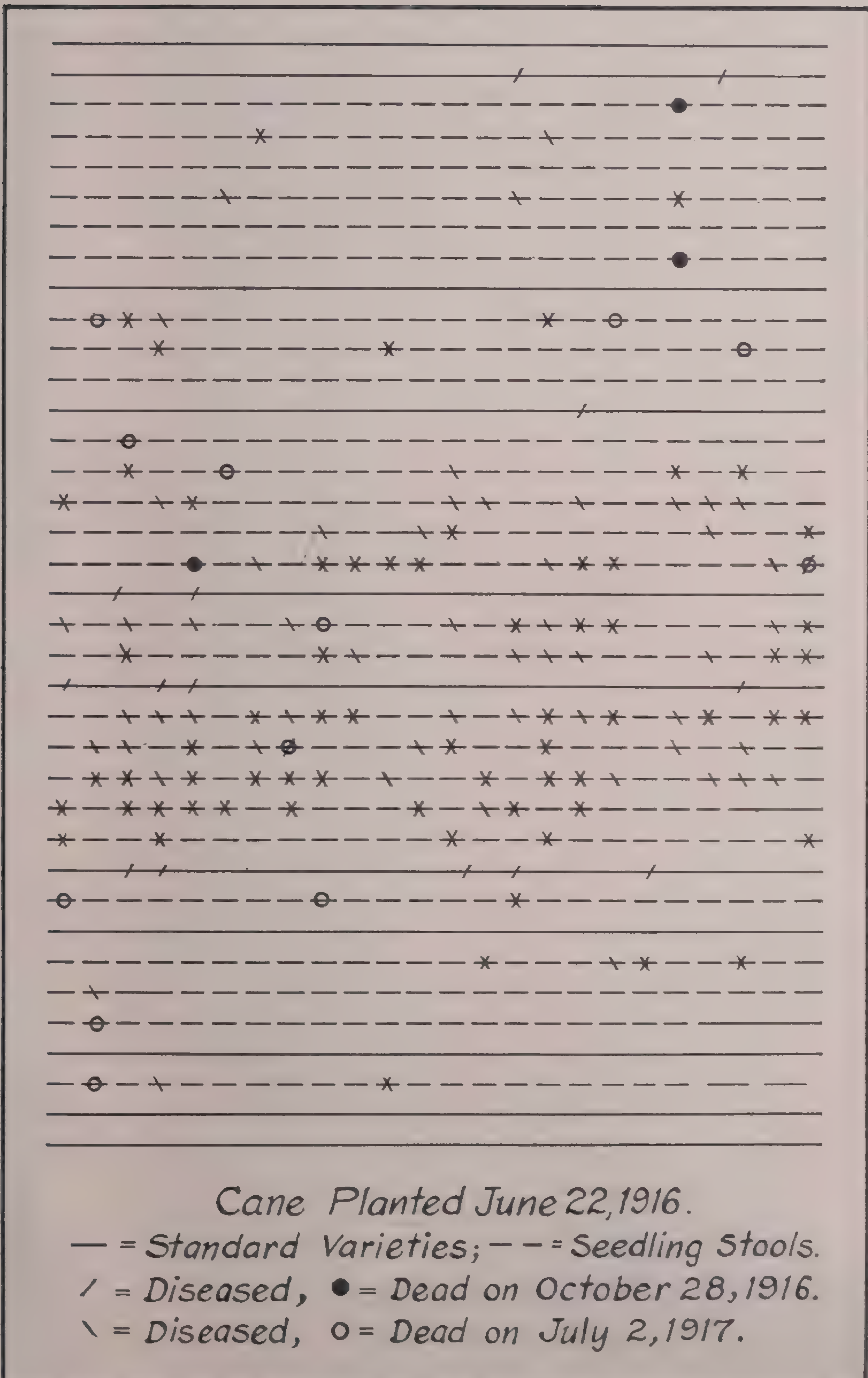


Figure 6.—Distribution of Mosaic in field. Chart showing the occurrence of diseased and dead stools in a block of seedling canes on the two dates indicated. The solid lines represent rows of standard canes grown from cuttings.

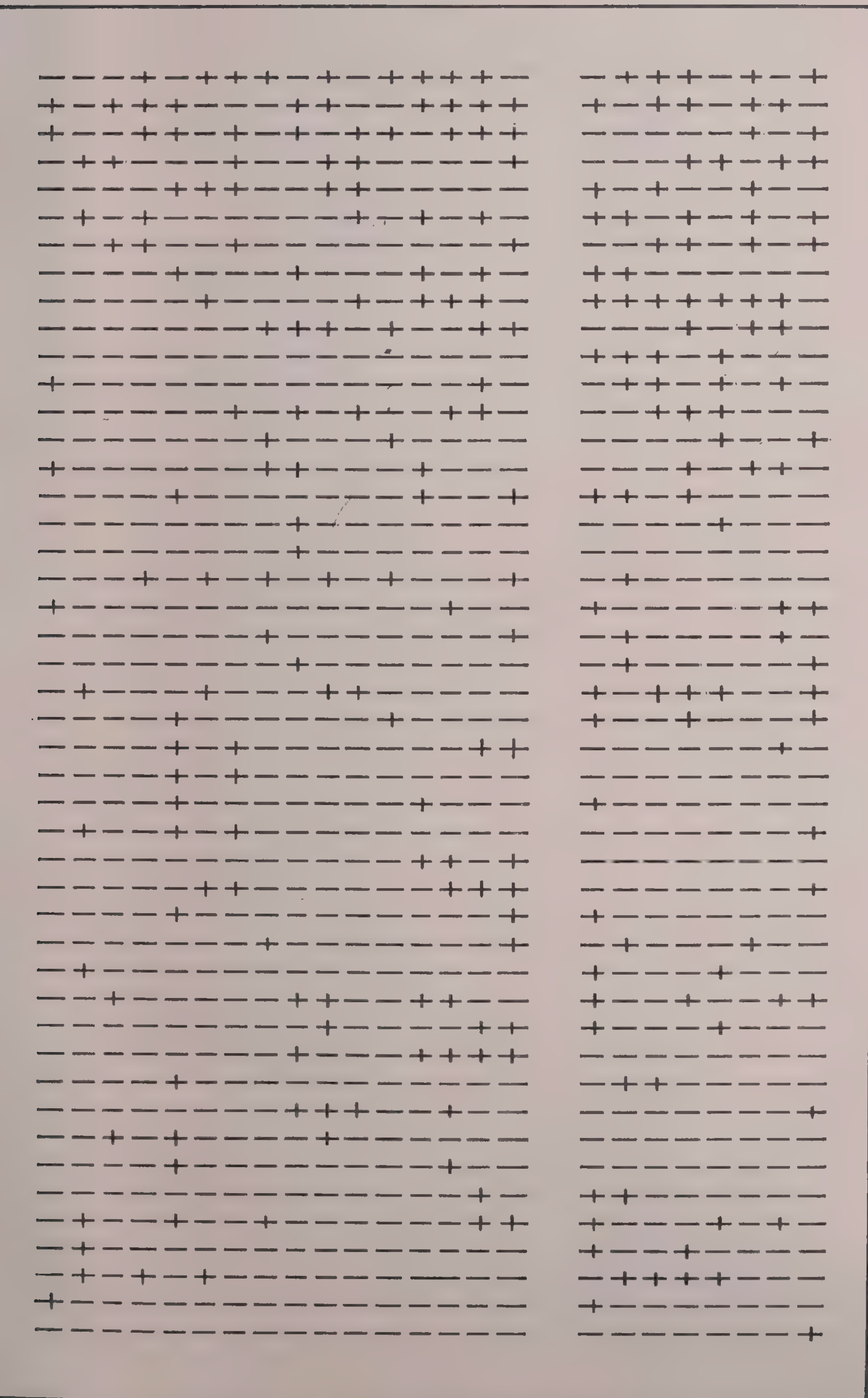
As an eye or lateral bud is differentiated in the axil of each leaf at the growing-point it is in such a position that it can scarcely escape invasion by the causative agent, and it is a rule, subject to very few exceptions, that when a leaf is mottled the corresponding eye will contain the infection and any shoot developed therefrom will show the disease.

The progress of the disease in individual cane stools would also seem to indicate that the causative agent does not migrate to any extent, if at all, through the tissues of the stem from one shoot to the next, but that infection normally spreads by the aerial route from shoot to shoot even in the same stool.

In a plant crop of H 146 three adjacent stools contracted Mosaic, the disease eventually extending to every shoot in the stools. This cane was harvested in February, 1913, and immediately ratooned. Every shoot arising from these three stools was healthy and remained healthy until June 23, 1913, when the stools were dug out. All of the primary shoots in a fourth stool of the same planting contracted Mosaic, but a secondary shoot (sucker), starting considerably later than the primaries, did not contract the disease and was healthy when the plant crop was harvested. Ratoon shoots from this stool came up strong and on June 1, 1913, the stool consisted of 19 shoots and all healthy.

In January, 1911, healthy cuttings of Lahaina cane, secured from a field in which Mosaic did not occur, were planted in a forty-foot row paralleling a row in which were canes already carrying Mosaic. The young shoots growing from these healthy cuttings were carefully watched and as soon as one showed Mosaic it was labeled and the date recorded. During March, April and May the disease spread rather freely among these canes, but new cases were very infrequent between June and October, and between October and the following April, when the canes were cut, no new cases developed. On October 11, 1911, the labeled sticks, of which there were then 35, were carefully inspected and all found to show the characteristic blotches on their youngest leaves, but on that date it was noted that in many cases the youngest leaves did not show as many blotches as did the preceding leaves on the same shoots. During November and December, 7 of the 35 shoots threw off the malady entirely and produced leaves on which no blotches at all occurred, and these seven shoots continued to produce healthy leaves up to the time that the canes were cut. Cuttings from five of these sticks which had recovered were planted. All top cuttings from the portions carrying healthy leaves gave rise to healthy shoots, but cuttings from the older portions which had carried diseased leaves gave rise to diseased shoots.

The observations on Mosaic in H 146 and Lahaina recorded above help to explain some of the anomalous conditions noted in the field in connection with this malady. Our records show that in several fields the disease has been far less prevalent in a ratoon than it was in the preceding plant crop from the same stools. A census of shoots in a small area, taken in April, showed 31% diseased, while a census of the same area in September showed only 18% diseased, but an increase of over 400% in the total number of shoots in the area.



igure 7—Distribution of Mosaic in field. Chart showing the occurrence of diseased stools in two blocks of seedling canes. Census taken about five months after planting out.

LOSSES DUE TO MOSAIC.

Any agency which effects a reduction in the amount of chlorophyll operated by a plant must cause a proportionate reduction in the carbon-assimilating power of that plant. It should, therefore, be held axiomatic that Mosaic lessens the sugar manufacturing capacity of the cane plant.

When Striped Tip contracts Mosaic the affected shoots are stunted and distorted to a degree which renders them utterly worthless, as can be seen in text figure 3. Diseased shoots of Yellow Caledonia are also stunted and otherwise injured to an extent that renders them of little value. That Mosaic causes very serious losses in these two cane varieties is, therefore, too evident to be disputed. Certain other much favored cane varieties, such as Lahaina and Demerara 117, however, although very susceptible to the malady, carry it without displaying any conspicuous evidences of injury, and this led many local growers to contend that the disease caused no losses at all in these varieties. A great many seedling canes also stood up so well under the disease that they were classed in the same category with Lahaina and D 117.

This matter was made the subject of careful investigation, and evidence was obtained from twenty-three varieties which showed that none of them could carry the disease without showing a loss in sugar. We quote below from our report [1] on an experiment in which Lahaina cane was used.

> Lahaina cane is very susceptible to Yellow Stripe disease, but when afflicted with this malady it still produces sticks of good size and quality. In fact it appears to stand up so well under the disease that some observers have expressed the opinion that it really suffers no harm.
>
> The experiment described herewith was designed to show the comparative yields obtained from canes grown from healthy and diseased cuttings respectively. The canes from which the cuttings were taken for this experiment were grown at the Pathology Plot in Honolulu, receiving uniform treatment. No fertilizer whatsoever has been applied to the soil at the Plot since it was taken over as virgin soil.
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> The experiment, planted in eight 80-foot rows, was arranged as follows:
>
> Rows 1 and 8, outside, blanket rows.
> Rows 2, 4 and 6, cuttings from healthy canes.
> Rows 3, 5 and 7, cuttings from canes having Yellow Stripe disease.
>
> Two cuttings of three eyes each were taken from the top of each stick. All the cuttings were carefully inspected to see that each had three perfect eyes. Sixty cuttings, thirty top and thirty second, were planted in each row; the top and second cuttings being similarly placed in each row.
>
> The cuttings from sound and diseased canes sprouted equally well and gave, what appeared to be, a uniform stand of cane. Not a single stick tasseled in the experiment during the winter of 1912–13, but most of them tasseled in November, 1913.
>
> The cane was cut during the last week in February, 1914, giving the following results:

[1] The Hawaiian Planters' Record 10:320-321, 1914.

From healthy cuttings, rows 2, 4 and 6—

Healthy canes	430	weighing	3991.0	lbs.
Diseased "	81	"	693.5	"
Undetermined canes	137	"	887.5	"
Total millable "	648	"	5572.0	"
Dead canes	187	"	553.5	"

From diseased cuttings, rows 3, 5 and 7—

Healthy canes	3	weighing	28.0	lbs.
Diseased "	335	"	2683.5	"
Undetermined canes	75	"	387.5	"
Total millable "	413	"	3099.0	"
Dead canes	210	"	534.5	"

Juice samples were obtained by grinding the cane from corresponding sections in the centers of two rows. The analyses were as follows:

From healthy cuttings,	Brix 20.3,	Sucrose 19.07,	Purity 93.9
" diseased "	" 20.1,	" 19.11,	" 95.1

The yields per acre computed from the above data would be:

From healthy cuttings	101.13	tons	cane,	14.98	tons	sugar
" diseased "	56.24	"	"	8.43	"	"
Difference	44.89	"	"	6.55	"	"

When comparing these yields it should be noted that 12% of the canes from healthy cuttings became diseased during their growth, so that the yield from healthy cuttings was thereby somewhat reduced.

The results of this experiment leave no room for doubt as to the deleterious effects of Yellow Stripe disease upon Lahaina cane. Stools from diseased cuttings yield only 60% as much sugar as stools from healthy cuttings.

Control Measures.

All measures for the control of Mosaic should be undertaken with a view to the ultimate elimination of the disease from any area under consideration. The method of procedure to be adopted must depend upon the conditions existing when these measures are instituted. If the disease occurs sparingly and in a few spots only it will usually be advisable to eliminate it at once by removing and destroying all affected canes, but if it is generally distributed throughout the area or any considerable part of it, rogueing is both ineffective and expensive. Under such conditions it is advisable to handle the area as a whole and interpolate corrective measures only in the regular routine of plantation operations. In other words the diseased cane should be allowed to stand until the grinding season opens, when it should be cut and sent to the mill. Then steps should be taken to eliminate the disease from the area and to prevent its appearance in the next crop on the same land. To accomplish this it is necescessary to plow out all of the old stools and insure their destruction before the field is replanted. Where it is possible to fallow and pasture the area this is easily accomplished, but in cases where the area must be replanted within a few months the desired result is not so easily obtained. It has been often observed that in fields where infected stools have been plowed out and the field

replanted soon after, many diseased shoots spring from portions of the old stools which have been buried, and these diseased shoots are of course a source from which infection may again spread through the entire area.

On any plantation or in any district where Mosaic occurs the greatest care should be exercised in the selection of cane from which cuttings are to be taken for planting, regardless of whether these cuttings are to be planted in an area where the disease has occurred or in an area where it has never occurred. If possible cuttings should be taken only in fields where the disease does not occur. If such fields are not available for seed-purposes cuttings should be taken only from healthy canes which are as far removed from diseased canes as existing conditions will permit. Of course it should be possible to get a stand of perfectly healthy cane from cuttings taken from healthy shoots which are mingled with diseased shoots in the same or adjacent stools, but when this is attempted on any scale at all a considerable number of diseased shoots usually spring from the cuttings so selected. This may be due to error on the part of the one making the selections or it may be due to the inclusion of the tops from canes which have but recently become infected, but do not display any blotches on their leaves, or it may even be due to the selection of some canes which have thrown off the disease but still retain the infection in some of their dormant eyes. At any rate in field practice it is always advisable to obtain cuttings for purposes of propagation from healthy canes which are as far removed from diseased canes as existing conditions will permit.

When Mosaic has become widely distributed in any area and shows ability to spread rapidly in the varieties under cultivation it is often advisable to replace the susceptible varieties with more resistant ones; in fact this has proven the only practical method of procedure on certain areas in Hawaii. Here no plantation can afford to replant its entire area in a shorter period than ten years. If the disease has become generally disseminated throughout a plantation it will spread from old fields through each replanted field within a year or two after planting if a susceptible variety is used, and thus very little headway can be made towards eliminating the disease even though cuttings are taken from healthy plants only. The elimination of Mosaic from certain areas where Lahaina cane was badly diseased has been effected by replacing it with Striped Mexican and equal success has resulted in other places where Striped Tip has been replaced with Demerara 1135.

The resistant varieties may not be as good canes as are the susceptible varieties when the latter are at their best, but healthy canes of the resistant varieties are certain to give better returns than diseased canes of the susceptible varities. After the disease has once been eliminated from a plantation by the use of resistant varieties the susceptible varieties may then be re-introduced by bringing in cuttings from some area free from Mosaic.

Summary.

1. Mosaic of the sugar cane is an infectious disease.
2. Cuttings from diseased sticks almost invariably give rise to diseased shoots.

3. The appearance of light blotches on the leaves is the primary and critical symptom of Mosaic and the only one on which a diagnosis should be based.
4. No cane variety grown commercially in Hawaii is immune to Mosaic, but a few varieties are very resistant to the disease.
5. Some cane varieties are very sensitive to Mosaic, being rendered quite worthless when attacked, while others stand up well under the disease, but no cane variety can carry Mosaic without suffering injury which entails a loss in sugar to the grower.
6. Mosaic may be controlled by
   A. the use of resistant varieties, and
   B. the use of cuttings from healthy canes only for purposes of propagation.

SEREH.

Sereh is the Javanese name for lemon-grass *(Andropogon Schoenanthus* L.) and this name was applied to the cane disease in question because the disease arrested the growth of the cane stool, converting it into a bushy tuft of leaves somewhat resembling the tufts of lemon-grass. This peculiar disease of the sugar cane suddenly appeared in West Java in the early eighties of the last century, and spreading rapidly through the fields of the island created one of the most serious plant disease epidemics on record.

Since its appearance in Java the disease has been reported from Borneo, Sumatra, Malakka, India, Mauritius, Australia, Fiji, Formosa, and Hawaii. It is reasonable to suppose that the disease actually spread to Borneo, Sumatra, and other islands of the Malay Archipelago, but we know that some of the maladies which have been diagnosed as Sereh in Australia, Fiji, and Hawaii were in reality other diseases. The cane growers of the West Indies were at one time inclined to believe that Sereh was present in their fields, but after consulting Dr. Went, who had had much experience with the disease in Java, they concluded that their cane troubles were not to be ascribed to this disease.

Promiscuous importation of canes from Java since Sereh reached its maximum distribution in that island has been practised by cane growers in several countries far removed from Java. Considering our inadequate knowledge of the disease and knowing its great potentialities for doing harm, such procedure is quite unjustifiable.

The literature dealing with Sereh is very extensive, and we shall not attempt to give an exhaustive review of it here. The best as well as the most available sources of information are the well known books of Wakker and Went,[1] and Krüger.[2] Here are to be found careful descriptions of the disease, good illustrations of serehed canes, and a concise resumé of all the early literature.

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[1] Wakker, J. H., en Went, F. A. F. C. De Ziekten van het Suikerriet op Java. 1898. (De Serehziekte, 76–98.)

[2] Krüger, W. Das Zuckerrohr und seine Kultur. 1899. (Die Serehkrankheit des Zuckerrohrs, 423–435.)

A comprehensive abstract of Krüger's discussion of Sereh appeared in Sorauer's [1] Handbuch.

The descriptions of Sereh which have been printed in the English language are very brief and not accompanied by illustrations. Dr. Benecke [2] supplied the first, which has been twice quoted by Deerr.[3,4] Dr. Kottman [5] contributed a very interesting discussion of Sereh and the various theories as to its cause. Dr.

Figure 8 –*Sereh.* A photograph used in illustration of the disease by Wakker and Went in their book "De Ziekten van het Suikerriet op Java."

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1 Sorauer, P. Handbuch der Pflanzenkrankheiten. (Die Serehkrankheit des Zuckerrohrs, 1:686–690.)

2 Anon. The "Sereh" disease in the cane. The Sugar Cane, 22:360-361. 1890. A short note containing "Symptoms of the 'Sereh' disease as given by Dr. Benecke."

3 Deerr, Noel. Sugar and the sugar cane. 1905. (Sereh, 73-75.)

4 Deerr, Noel. Cane Sugar. 1911. (Sereh, 145-147.)

Went [1] supplied the latest and most concise statement on the subject. His views are of particular interest and value as they represent the matured conclusions of one who worked with Sereh when it was at its worst in Java and has followed it through every phase of its later history. A good resumé of the early work on the disease has been rendered into English through the translation of Sorauer's Handbuch.[2]

Sereh has probably received critical study at the hands of a greater number of competent pathologists than has any other disease peculiar to a cultivated plant of the tropics. These investigators have disagreed among themselves as to the cause of the malady, and as no one has been able to bring forward conclusive proof to substantiate his findings the causative factor still remains obscure, or at least insufficiently demonstrated.

The sudden appearance and rapid spread of the disease clearly indicated to many investigators that it was caused by some parasitic organism, but failure to demonstrate the responsible parasite permitted the exploitation of other theories regarding the primary cause.

Bacteria, root-destroying fungi, and nematodes were the organisms held responsible for the malady by the advocates of the parasitic theory; the strongest evidence being built up by those who ascribed the disease to bacterial activity. Unfavorable climatic conditions induced by deforestation of the mountains, drought, excessive moisture, improper cultivation and fertilization, physiological disorders, derangement of the enzymatical functions, and degeneration were the explanations seriously advanced by those disputing the parasitical nature of the causative factor. Finally Van der Stok included Sereh with Gele Strepenziekte, explaining both as but cases of prolific sporting in a hybrid race of cultivated plants.

Diagnosis.

When Sereh first appeared in Java the Cheribon cane was the one standard variety grown throughout the island, and the early studies and descriptions of the disease relate almost entirely to this one variety. When afflicted with Sereh the Cheribon cane shows the following symptoms to a greater or less extent:

1. Inability to grow; the majority of shoots remaining short and stunted.
2. Vascular bundles in sticks colored red, due to the presence of a red, gummy substance in the vessels.
3. Pronounced growth of adventitious roots under the leaf sheaths from many or all of the nodes on diseased sticks.

If affected with Sereh the Cheribon cane always shows the first symptom mentioned above. The second symptom can usually be detected by careful dissection of the older sticks, but the third symptom may or may not be in evidence and consequently is of little diagnostic value. In later years the seedling cane 247B has replaced Cheribon to a very large extent as the leading commer-

[1] Went, F. A. F. C. Does the Sereh disease exist in the West Indies, more especially in Trinidad? West Indian Bulletin, 12:554–558. 1912.

[2] Sorauer, P. Manual of plant diseases. (The Sereh disease of the sugar cane, 1:692–696.)

cial variety in Java. This cane is nearly or quite as subject to Sereh as is the old Cheribon, but the disease does not cause a stunted growth in this variety, affected shoots growing as rapidly and to the same length and diameter as healthy ones.

The diseased sticks are always made conspicuous, however, by a copious growth of adventitious roots from their aerial nodes (text figure 9), and red vascular bundles are usually numerous and conspicuous when the sticks are split open. (Plate 3, figure 2.) An additional symptom shown by diseased sticks is a lack of sap in the cells occupying the center of the internodes. As a result this central tissue is white and much resembles an axillary strand of pith.

The following peculiarities displayed by serehed canes may be made use of in testing suspected canes:

1. The disease is always transmitted through cuttings, the symptoms being more pronounced in each succeeding generation.

2. The upper eyes on serehed sticks which have been topped remain dormant, i. e., they do not grow out into lateral shoots as they always do on healthy sticks.

Fig. 9.—*Sereh.* A stick of the Java seedling cane 247 B showing copious growth of adventitious roots.

Sereh in the Field.

Prior to the appearance of Sereh and during the first years that it was present in Java, the cane growers in some districts were in the habit of taking one or more ratoon crops. Very early in the history of the malady, however, it became evident that a cane stool once attacked by the disease never recovered; that the morbid symptoms increased and accumulated with age; that stools which produced a few moderate sized sticks in the plant crop would yield no sticks at all in the succeeding ratoon; and that many of the stools which were apparently healthy in the plant crop would be badly diseased in the ratoons. Consequently, the growing of ratoons was abandoned.

The early students of Sereh describe certain symptoms which apparently develop only in the ratoon crop after the stools have been long diseased. Several investigators mention that secondary parasitic organisms are wont to congregate in the serehed stools and it is quite probable that some of the symptoms attributed to Sereh were in reality caused by these secondary parasites. In 1890 Dr. Benecke wrote: "In reality they now call every diseased appearance of the sugar cane 'Sereh' and thereby cause the greatest confusion."

When the writer visited Java in 1911 for the purpose of studying Sereh, he found that his studies must be confined to the disease in the plant crop as no ratoons were available. The symptoms for diagnosis as previously given and the following description of the disease in the field are consequently based on a study of the disease in plant cane only.

When a plant of Cheribon cane is attacked by Sereh the one conspicuous effect of the disease is the early suspension of growth in all shoots which arise from the stool. A shoot will push up out of the ground in the usual manner, maintain a normal growth for a time and then for no apparent reason stop growing. This procedure will be repeated by shoot after shoot, each in turn ceasing to grow at an early stage; thus the stool is converted into a bunch of short shoots of no commercial value. In such a stool the various shoots usually arrive at different lengths before ceasing to grow and one or two may produce sticks several inches in length. The serehed stools in fields of Cheribon cane in Java are usually of this type, each stool consisting of one or more long shoots surrounded by a cluster of short grassy shoots. The stools shown in text figures 10 and 11 are typical serehed stools of Cheribon.

The occurrence of red bundles in the stem is the symptom of Sereh on which the most stress has been laid by all students of the malady. Strange to say, however, this symptom is not readily detected in serehed sticks of Cheribon until they are at least ten months old. Even in sticks of this age which were grown from cuttings taken from serehed canes, red bundles were found to be very scarce and inconspicuous. Such red coloring as occurred in these sticks was confined almost entirely to such portions of the bundles as lay within the nodes, the color not extending into the internodes to any distance.

An excessive growth of adventitious roots on the aerial portion of a stick is another symptom which is of little aid in the detection of Sereh in Cheribon, for in the majority of diseased stools such roots are not present to an abnormal extent.

When it comes to identifying Sereh in the seedling 247B, the various symptoms have entirely different values from those indicated above for the disease in Cheribon. In the first place, diseased stools of 247B produce just as many sticks and just as large sticks as do healthy stools, so "Sereh" or "lemon-grass" disease does not in any way describe the malady in this variety. On the other hand serehed sticks of 247B begin to throw out adventitious roots from their aerial nodes when quite small and it is by this symptom that they are most easily detected in the field. The serehed stick of 247B illustrated in text figure 9 shows a luxuriant growth of adventitious roots. This is an extreme case, it is true, but diseased sticks always show enough adventitious roots so that they can be readily distinguished from the healthy ones by this character.

Red vascular bundles are always numerous and conspicuous in serehed sticks of 247B and in this variety constitute the one critical symptom on which a certain diagnosis can be based. The red material seems to be concentrated in the nodes and from these points extends up and down into the internodes above and below. Not infrequently a bundle is red throughout the length of an internode, and occasionally one may encounter a bundle which continues red uninterruptedly through two or three internodes. The colored drawing, Plate 3, figure 2, shows a serehed stick of 247B in longitudinal section.

Figure 10.—*Sereh.* The subject for this illustration was selected by Miss G. Wilbrink as a typical case of the disease in Cheribon.

While a serehed stick of 247B may be as large as a healthy stick of the same age, it is always very much lighter in weight and yields far less in juice and sugar. The reason for this lies in the fact that the parenchyma in the internodes, which should be filled with sweet cell-sap, is in reality very poorly supplied with this commodity. In a serehed stick the parenchymatous tissue occupying the axis

Figure 11.—*Sereh.* The stool of cane in the foreground is afflicted with the disease. It is ten months' old Cheribon. Healthy cane of the same variety which has made a normal growth can be seen in the background.

of each internode early loses all the sap from its cells and assumes a dry, pithy consistency. In the older internodes near the base of the stick, this pithy central tissue often breaks down, leaving a cavity of considerable size.

Having enumerated the symptoms as displayed by Cheribon and 247B

when affected with Sereh, we have covered the full range of symptoms by which the disease may be detected in any variety of cane grown in Java. Red gum in the vascular bundles is the one symptom, however, most relied upon by the Java cane pathologists for the detection of the disease in one and all varieties. When examining canes in the field for Sereh it is their custom to cut into the side of each suspected stick at a node in search of red bundles.

One symptom, often cited and figured by the early students of Sereh, is sprouting of the eyes on the lower nodes of diseased sticks, thus forming numerous lateral shoots or lalas which add to the "lemon-grass" appearance of the stool. This might very naturally be expected to occur in a diseased stool

Figure 12.—*Sereh.* The cane to the left of the stake is Cheribon grown from cuttings taken from plants afflicted with Sereh. The cane on the right of the stake is of the same age as the Cheribon but grown from cuttings taken from healthy plants.

of Cheribon, for the prolific shooting of the eyes below ground is a conspicuous symptom of Sereh in this variety. The writer did not meet with a single shoot, however, in which lalas had developed on the serehed sticks. It is a symptom that probably appears at a relatively late stage of the disease and in certain varieties only.

In late years it has been shown that failure to produce lateral shoots from its upper eyes by a stick from which the leafy top has been removed is direct evidence that that stick has Sereh. This interesting fact first became evident in the production of "keberi bibits" on certain plantations. In addition to using

top "bibits" or cuttings for planting, the Java planters sometimes find it necessary to plant "body-seed" from nearly mature cane. As the eyes on body-seed start rather slowly if such seed is simply cut from the stick and placed directly in the ground, they often "sprout" these eyes on the standing cane before making the cuttings. These sprouted cuttings are their "keberi bibits" and they secure them in the following manner:

The tops of standing canes are cut off and converted into the usual top-cuttings. The naked sticks are then allowed to stand in the field and in a short time the upper eyes grow out into short, lateral shoots. When the larger shoots have attained a length of ten or twelve inches they cut off the upper four or six joints and leave the sticks to produce another crop of shoots, when a second shoot-bearing section is removed. This operation is repeated until the entire sticks are used up or until they refuse to throw out any more shoots.

The shoot-bearing portions, after being removed from the sticks in the field, are carefully cut across through the middle of each internode, thus isolating each node with its subtended shoot. These short, sprouted but rootless cuttings are then set upright in the ground with the upper cut end exposed. The leaves are cut back to reduce transpiration until the roots have started sufficiently to keep up the water supply in the tissues. Such are the "keberi bibits" of the Java sugar planter and such is his method of obtaining them.

Now in the process of obtaining keberi bibits on certain plantations it was noticed that the upper eyes on some canes failed to shoot after the tops had been removed. An examination of such canes showed that almost without exception they displayed the critical symptoms of Sereh. The conclusion became general, therefore, that the use of keberi bibits offered a reliable means of selecting seed from healthy canes in fields where Sereh existed. The Sugar Planters' Experiment Station in East Java put this matter to a rigid test in careful experiments in the field. They reported the results and conclusions arrived at as follows:[1]

> "1. That all of the stalks on which the eyes had not budded were Sereh-sick.
>
> "2. That the stalks on which the eyes had only partly budded were generally Sereh-sick.
>
> "3. That of the stalks on which all the eyes had budded less than 1% were Sereh-sick.
>
> "Which proves that in planting with keberi bibits one eliminates the Sereh-sick stalks to a great extent if one takes care to use only bibits of those stalks on which all of the undamaged eyes have budded."

Control of Sereh in Java.

After a few years' experience with Sereh the Java planters came to recognize certain definite results which they could expect to obtain with it in their fields. If they planted cuttings from serehed sticks they got only badly diseased stools. If they planted cuttings from healthy sticks only they got a sufficient

[1] Jaarverslag, 1910, Pg. 63 and following.

number of healthy stools to make a relatively good crop. If they ratooned their fields the disease was sure to take so many additional stools in the second crop as to render it a failure.

When the planters became thoroughly acquainted with these facts they first abandoned the practice of ratooning and then endeavored to secure bibits or cuttings from healthy stools only for each succeeding planting. Painstaking selection of healthy canes for seed has been the chief means whereby the Java planters have maintained their sugar production at a high level.


Figure 13.—*Bunch-top*. A cluster of deformed buds resulting from the abortion of an inflorescence.

At a relatively early date in the history of Sereh it became evident that the disease confined its operations to the lowland fields and did not flourish at high elevations. This at once indicated a method whereby healthy canes could be obtained for purposes of propagation, and the Java planters have elaborated a system of seed-cane production somewhat as follows: Primary nurseries or "grandmother-fields" are located at elevations of 5000 to 6000 feet. The cane grown in these primary nurseries is cut when six to eight months old and converted into bibits or cuttings which are employed for planting secondary nurseries or "mother-fields," at 2000 to 2500 feet elevation. The cane produced in the secondary nurseries is in turn used as "seed" for planting a third series of nurseries, the "daughter-fields" or "export-nurseries," which are usually located at about 1000 feet elevation. From the "export-nurseries" cuttings are "exported" to the plantations where they are employed for planting the lowland fields. Strict inspection of all canes is practiced at each and every nursery and any sticks showing symptoms of Sereh are promptly discarded. Cane "seed" obtained by this rather tedious process is quite free from Sereh and fields planted with it remain sufficiently free from the disease so that seed can be safely cut from them for planting the succeeding year.

In fact, on plantations where careful selection is practiced it is only necessary to bring in mountain-grown seed every four or five years to keep the fields reasonably free from Sereh. From this it is not to be inferred that every plantation in Java uses seed grown in mountain nurseries, for we were reliably informed that such was not the case, some plantations having always been able to maintain a proper stand of cane by selecting seed-cane from their own fields.

In the early days of Sereh, great hopes were placed on varieties of cane imported from foreign countries, but all of the good sugar producing varieties

imported proved to be no more resistant to the disease than the standard varieties of Java had been.

The growing of seedling canes has seemed to offer the greatest promise of affording a solution to the Sereh problem in Java. There is always the possibility that a cane may be secured in this manner that is remarkable as a sugar producer and at the same time immune to Sereh. They have obtained seedlings showing one or the other of these characters to a marked degree, but they have not as yet secured one posesssing both characters. They have, however, obtained several seedlings which are superior to the old standard varieties under existing conditions, and these seedlings now occupy the greater part of the lands under cane in Java.

Figure 14.—*Bunch-top.* One of the many forms resulting when cane inflorescences revert to vegetative growth; in this case a cluster of thin narrow leaves devoid of midribs.

The Chunnee cane, a very slender reed-like variety, imported from India, proved to be immune to Sereh. It was at once hailed as the possible progenitor of the desired immune cane. Under the direction of Dr. Kobus, an enormous number of seedlings were obtained by crossing Chunnee with other and larger varieties, Cheribon being the favorite second parent. The results obtained were not equal to expectations, however, as few creditable canes were secured and many of these were subject to Sereh. This is true of No. 228 P.O.J., which is one of their very best Cheribon x Chunnee seedlings.

Sereh in Hawaii.

In the paragraph quoted below Dr. Walter Maxwell[1] records the early introduction of Sereh into Hawaii:

> "At this place we urge the absolute necessity for all varieties introduced into this country being sent first to the station for trial. This is to guard against the introduction of disease. As an example, this year an excellent variety was brought from Fiji by Prof. Koebele, and sent to the station for trial. It came up and grew well, being full of promise. When seven months old, abnormal symptoms developed. Prof. Koebele was called in to inspect and found some symptoms of *Sereh disease*, but not conclusive. Our personal comparison of the symptoms with the notes on the disease by the great authority, Dr. W.

[1] Directors' Report for the year 1900.

Krüger, made it evident to us that we had a case of the Sereh scourge. We dug up and burnt the whole plat, and sent out to two plantations, who had obtained a few sticks, asking them to also utterly destroy the same. The Association should seek to have it made imperative for all introduced varieties to go for trial at the experiment station, and it is advisable that the station shall in future withstand all solicitations for seed of new varieties until these have furnished a mature crop, and are eligible for a clean bill of health. The variety in question is the 'Malabar.' We believe it is originally a Java variety; the name following probably from the village or mountain of that name in Java."

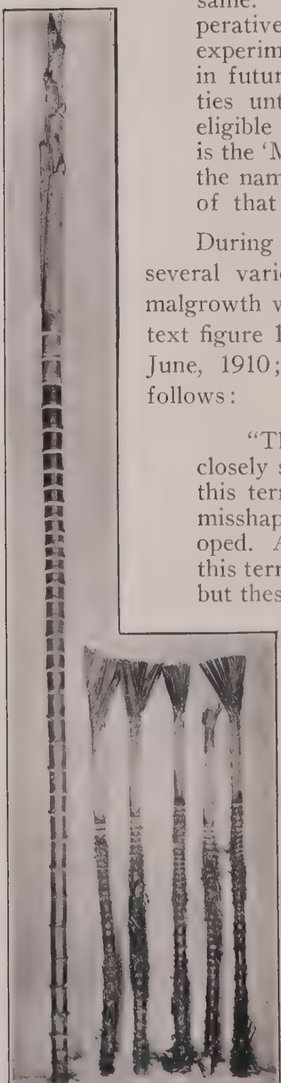

Fig. 15.—*Dwarfs.* Stunted growth in Yellow Caledonia simulating Sereh. The sticks are all of the same age but only the one on the left has made a normal growth.

During the spring of 1910 a peculiar malgrowth was noted on several varieties of cane, on widely separated plantations. This malgrowth was dubbed "bunch-top" and the cut here reproduced as text figure 13 was published in The Hawaiian Planters' Record for June, 1910; the specimen illustrated being there described as follows:

"The top of the stick was covered by a bunch of closely set, scale-like leaves. A longitudinal section showed this terminal bunch of leaves to be in reality a cluster of misshapen buds between which no internodes had developed. A great many roots had grown out from the base of this terminal bud-cluster and also from the first node below, but these were cut away before the sketch was made. The first three nodes below the bud-cluster were devoid of eyes, while the eye of the fourth had started to grow."

In the note quoted from above we classed this and similar specimens as mere freaks, at the same time distinguishing between *galls* and *freaks* as follows:

"Galls are malgrowths induced by extraneous causes, such as improper nutrition, mechanical injury, and attacks of parasites. They are strictly pathological growths.

"Freaks are malgrowths following a chance derangement of that internal mechanism of the plant which determines its growth; the mechanism slips a cog, we might say, and erratic growth follows."

Now while we held this "bunch-top" to be but a freaky growth of the cane plant, still it occurred with great frequency in the cane fields during 1910, becoming particularly noticeable during June and July, for the buds and leaves in the bunch-tops grew out into close tufts of short shoots and strap-shaped leaves, often extending well beyond the normal leaves as a conspicuous brush (text figure 14). As the shoots and leaves in these bunch-tops were

always small and closely crowded they presented an appearance not unlike a bunch of lemon-grass and it was suggested that they might be malgrowths induced by Sereh.

Figure 16.—*Dwarfs.* Stunted growth in Yellow Caledonia simulating Sereh. Note the copious growths of adventitious roots and compare with figures 9 and 17.

During 1909 and 1910 the Striped Tip variety of cane, over considerable areas in the Hamakua district of Hawaii, suffered the marked dwarfing which was at that time known to be the symptom of but one cane disease, Sereh.

Thus we had first a record of the introduction of Sereh into Hawaii from

Fiji, then a peculiar and recurring malgrowth, bunch-top, resembling a bunch of lemon-grass, and finally a malady which caused a marked dwarfing of the cane, a symptom always cited as most characteristic of Sereh. As this combined evidence was arousing considerable apprehension in Hawaii, it was decided that the writer should visit Fiji and Java and make a thorough study of the cane diseases in those countries in order to correctly interpret these suspicious symptoms displayed by canes in our own fields. The results of these investigations abroad, and further studies of the local cane maladies, led to the following conclusions:

1. Dr. Maxwell was mistaken in his diagnosis, for Sereh has never been detected in Fiji. If the canes introduced from Fiji by Dr. Koebele were affected with some disease that stunted their growth, that disease was most probably Mosaic, but possibly Fiji disease.

2. Bunch-top is a malgrowth resulting through the abortion of an inflorescence or tassel. The growing point of the shoot reverts to vegetative growth after starting the development of an inflorescence, and a bunch-top is the result.

3. Dwarfing of the Striped Tip cane we soon correlated with the mottling of the leaves due to Mosaic, finding that this symptom was as characteristic of Mosaic in Striped Tip as it was of Sereh in Cheribon.

While we were thus able to nullify all of the evidence formerly held to support the view that Sereh was present in Hawaii, our studies of the disease in Java gave new significance to certain cases of stunted growth in cane which we had often observed but confidently ascribed to the combined effects of poor soil, poor culture, and adverse climatic conditions. These stunted stools have been observed in many parts of the islands, but are of most frequent occurrence in the Hilo district on Hawaii, where they have been found in several varieties of cane, but most frequently in Yellow Caledonia and Demerara 1135. As a rule the stunted stools occur in groups, or, in other words, all of the stools on certain patches are stunted. These patches are usually small, ranging from 10 to 50 feet in diameter, but we have seen a few patches of these stunted stools that covered approximately an acre of ground. The individual canes in these stunted stools often show all of the symptoms that are displayed by the Cheribon cane when it is afflicted with Sereh. Pronounced dwarfing of the shoots is accompanied by excessive development of adventitious roots, while red gum in the vascular bundles is often present in considerable amounts. (text figures 15 and 16.) If these stools occurred in a cane field in Java no one could possibly think of considering their malady any disease other than Sereh. During our investigation of these canes we took cuttings from the most seriously affected stools in every patch examined, and planted them in our quarantine field in Honolulu. Without a single exception these cuttings from diseased sticks gave rise to healthy stools, which produced a plant and two ratoon crops of sticks above the average in size and quite devoid of adventitious roots or red discolorations in their vascular bundles. This we consider conclusive proof that the stunted canes in question were not affected with Sereh. This conclusion has been substantiated by the behavior of the malady in later years, for it has shown no tendency to spread beyond the restricted areas where it first appeared.

During the winter of 1918–19 Mr. H. F. K. Douglas visited Hawaii on his way home to Holland from Java, where he had spent many years as manager of a sugar plantation. Upon seeing some dwarfed sticks of Yellow Caledonia similar to those shown in text figures 15 and 16, which were preserved in our museum, he promptly pronounced them typical serehed sticks. He was

Figure 17.—*Sereh.* A photograph used by Wakker and Went in their book, "De Ziekten van het Suikerreit op Java," to illustrate the excessive development of adventitious roots induced by Sereh.

later taken to various points in the Hilo district and shown the stunted stools in the field. These he declared showed all of the outward symptoms displayed by serehed stools of Cheribon, but he was of the opinion that the red discolorations were not evident in their vascular bundles to an extent corresponding to the external symptoms displayed.

When the history of this malady in our fields and the results obtained by planting cuttings from these stunted stools in good soil were brought to his attention, Mr. Douglas at once stated that the stunting of the cane was not due to Sereh.

SUMMARY.

1. Sereh is unquestionably an infectious cane disease, but the exact nature of the causative factor is unknown.
2. Sereh has no strictly unique symptoms, for all symptoms credited to it in Java are induced at times by other diseases in other countries.
3. The one symptom by which this disease can most often be detected is the presence of red gum at points in the vascular tissue of the stem.
4. Sereh causes a pronounced stunting of the stools in some varieties of cane.
5. A pronounced growth of adventitious roots from the aerial nodes of the sticks is often induced by Sereh.
6. Sereh is always transmitted through cuttings, the symptoms being more pronounced in each succeeding generation.
7. Sereh is controlled in Java by
    A. the use of resistant varieties,
    B. the use of cuttings from healthy canes only for purposes of propagation, and
    C. the abandonment of the practice of ratooning.

FIJI DISEASE.

Our first knowledge of the existence and nature of this unique cane disease was obtained in August, 1910, from photographs and material in formalin solution, sent up from Fiji in the hands of Mr. F. Muir by Mr. D. S. North of the Colonial Sugar Refining Company. In December of the same year the writer went to Fiji for the purpose of studying the disease in the field and securing properly fixed material for histological investigation. During 1911 and 1912 Mr. North spent nine months at our laboratories in Honolulu, devoting most of this time to the making of permanent histological preparations from the material collected by the writer. Upon his return to Fiji and Australia Mr. North collected and forwarded additional material for cytological study and also supplied much additional information regarding the distribution of the disease. It is obvious therefore that the following discussion of Fiji disease must be built in large part upon work done by Mr. North.

When the writer visited Fiji in 1910–11 it was considered as established that Fiji disease was endemic in Fiji. The disease was found on a wild, reedlike cane, *Veico*, and it was supposed to have spread from its native host into the fields of cultivated cane. At that time the disease was not known to occur outside of Fiji except in certain restricted districts in Australia to which it was supposed to have been carried from Fiji in cane cuttings.

In a letter dated October 21, 1914, Mr. North wrote from Sydney as follows:

> "A. M. Carne, one of our men, has recently returned from an expedition to New Guinea to collect new varieties. He worked mainly

Figure 18.—*Fiji Disease*. Plant cane seventeen months old affected with the disease. Of the variety in the foreground, every stool is dead. Of the variety in the background to the left, 80% of the stools are attacked, but too recently to be greatly damaged. Photographed by Mr. North.

mountainous, inland districts and travelled from the north side overland to Port Moresby. In all the districts he visited he found Fiji disease to be most prevalent, frequently finding difficulty in collecting any sound-looking cuttings of certain varieties. He also found one stool only of Yellow Stripe disease. He is thoroughly conversant with these diseases and brought back formalin specimens of them, which I now have, and their identity is beyond doubt."

This cane disease may have been carried from Fiji to Australia, but it certainly was never carried from Fiji or Australia to the wilds of New Guinea, and the conclusion is inevitable that it was indigenous to New Guinea. It is more than likely that the malady was actually endemic in New Guinea and migrated to Australia and Fiji with some of the many cane varieties which have, from time to time, been taken from New Guinea for purposes of cultivation on the sugar estates of Australia and Fiji.

Fig. 19.—*Fiji Disease.* Portion of a cane leaf seen from below, showing the ridge-like galls along the veins.

Fig. 20.—*Fiji Disease.* Two leaf galls enlarged twelve diameters.

Late in 1920 plant pathologists in the Philippines discovered Fiji disease in cane fields on the islands of Mindoro and Luzon. Mr. H. A. Lee has recently sent us carefully fixed material for cytological study and in this we find the pathological tissue characteristic of Fiji disease.

At the present time our information regarding the distribution of the disease in the Philippines is too meagre to warrant any conclusion as to the route by which it invaded this new territory. It is quite possible, to be sure, that it has, as in New Guinea, been present in small gardens of the natives in the interior of the islands from time immemorial and has only recently found its way into the large fields of cultivated cane. It is far more likely, however, that it has arrived by way of Australia or Fiji in cane varieties imported by the modern planters of the Philippines.

Diagnosis.

The one critical symptom by which Fiji disease may be recognized is the occurrence of elongated swellings or galls on the under surface of the leaves. These galls extend along the larger veins or vascular bundles and are, in fact, formed by the abnormal growth of the tissues comprising these bundles (text figures 19 and 20). Galls are produced in similar manner in the vascular bundles

Figure 21.—*Fiji Disease.* A stool of Badila in the last throes of the disease. Photographed by Mr. North.

of the stem and may be detected by splitting open the stick of an affected shoot,

Galls of this nature are not induced by any other known cane disease and consequently their presence on the leaves or in the stem of a cane plant may be accepted as conclusive evidence that that plant is afflicted with Fiji disease.

The most conspicuous symptom of Fiji disease to be noted in the field is a shortening and crumpling of the last leaves to unfold from the spindle (text figures 21 and 22). This peculiarity will attract the attention when one is still a

considerable distance from the affected cane. A diseased shoot may attain considerable length and be clothed with many healthy-looking leaves of the usual length and color, but of a sudden it loses the power to produce normal leaves, throws out a few bent and twisted stumps and then ceases to grow altogether. Some of the eyes may start, but the resulting lalas soon repeat the antics of the main stem. The stick may remain alive for months or it may soon die.

Figure 22.—*Fiji Disease.* This stalk has made its final effort to throw out leaves. Photographed by Mr. North.

When such a stick is examined the characteristic galls are usually to be found on most of the healthy-looking leaves which are not otherwise distorted and on all of the deformed, aborted leaves. These latter leaves look as though they had been burned or scalded before expanding, the injury destroying the upper half or two-thirds of the leaf blades, leaving short, crumpled stumps (text figures 22 and 23).

As indicated above, this abortion and distortion of the young leaves marks the culmination of the disease in a shoot and the last efforts of the growing point

to throw out leaves. A shoot may throw out leaf after leaf bearing galls, but otherwise normal, and grow on for months as though perfectly healthy; then of a sudden comes this final spasm in its growth, and it is done. The appearance of galls on the leaves is the first outward symptom by which the disease may be detected, but a cane may be hopelessly infected with it for months before any

Figure 23.—*Fiji Disease.* Distorted leaves from the tops of canes overcome by the disease. Photographed by Mr. North.

galls appear. The disease is therefore cumulative in the cane, the galls mark a well advanced stage of the disease, and the distortion of the apical leaves its final culmination.

Any cane stool which has once contracted Fiji disease must eventually succumb to the malady. No case is known where a cane shoot has recovered from,

or outgrown Fiji disease. Shoots grown from cuttings taken from diseased sticks invariably show galls on their first leaves.

The Causal Agent.

The writer based his first histological study of Fiji disease upon old galls from the material preserved in formalin solution sent up from Fiji by Mr. North. Sections showed that the pathological tissue of the gall was formed through the excessive multiplication of the cells on the phloem side of the vascular bundle. There was always considerable hypertrophy of the sclerotic tissue, but the strictly pathological tissue consisted of a large mass of pseudo-parenchyma. Each cell in this pseudo-parenchymatous gall-tissue contained in addition to a nucleus and cytoplasm a large, densely-staining body These foreign bodies were not uniform in size or shape, some being no larger than the nucleus in the same cell, while others would be a great many times the size of the nucleus which they accompanied. These bodies were usually vacuolate and appeared to have the structure of a dense protoplasm. While it was possible to differentiate structures resembling nuclei in some of these foreign bodies, the majority of them did not seem to possess any organ of this nature. As these foreign bodies appeared consistently in all of the pseudo-parenchymatous cells in the many galls examined, and as they closely simulated plasmodia, the writer looked upon them as organisms of this nature and in a short article published in The Hawaiian Planters' Record in October, 1910, announced this conclusion. The drawing here reproduced as text figure 25 appeared in this article. After the writer returned from Fiji he made many preparations from material which had been carefully fixed for cytological investigation. A large number of similar preparations made by Mr. North were also placed at his disposal. A study of these preparations strengthened his opinion that the foreign bodies were the individuals of a parasitic organism, but showed that the structure of this organism did not correspond to that of any known parasite, and he was, and still is, at a loss to know just where in the natural system this organism should be classi-

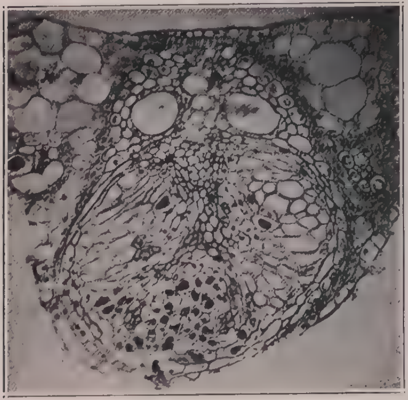

Figure 24.—*Fiji Disease.* Cross-section of a leaf-gall. The pseudo-parenchymatous gall-tissue with a dense body in each cell occupies a median position near the lower surface of the gall. Sectioned and photographed by Mr. North.

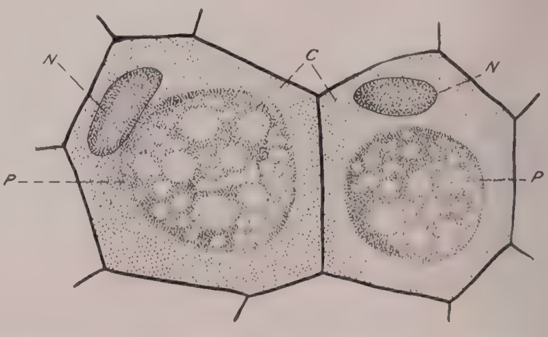

Figure 25.—*Fiji Disease.* Two cells from a stem-gall as seen in a thin section. P, foreign body, *Northiella sacchari*; N, nucleus, and C, cytoplasm of cane cell.

fied. In an unpublished manuscript the writer proposed the name *Northiella sacchari* for this unique cane parasite.

During the past year the writer has turned all of his material on Fiji disease over to Dr. L. O. Kunkel, who is now making a very careful cytological study of the organism. It is hoped that in the very near future Dr. Kunkel will be able to publish a critical description of this organism with details of its life history. At the present time we shall not go further than to say that it is always present in all of the stem and leaf galls of canes afflicted with Fiji disease; that it has been found in incipient galls only three millimeters below the growing point of the cane stem; and that in developing galls the individuals have been observed to divide simultaneously with the nuclei which they accompany.

Figure 26.—*Fiji Disease.* First ratoons at Nadi. The variety on the left is Childers Zigzag, a cane that has proven very resistant to the disease, while the variety on the right is Daniel Dupont, a cane that is very susceptible to the disease, but not easily killed out by it. Every stool of the Dupont was alive, but consisted of short leaves only, there being no sticks at all. Photographed by Mr. North.

Resistance and Susceptibility.

No variety of cane has yet been found which is immune to Fiji disease. Some varieties will grow eighteen months or more after infection before any galls appear. On other varieties, however, the galls appear soon after infection and the final spasm quickly follows. Thus it is that some of the more susceptible varieties are unable to make any growth at all in districts where the disease is epidemic. This is true of many of our favorite Hawaiian varieties including Lahaina, H 109 and D 1135.

At an early date it was found that the New Guinea cane, Badila, was exceptionally resistant to Fiji disease and that stools of this variety would remain alive and grow for months after galls began to appear in their leaves. Since Badila proved to be a superior cane in all other respects it naturally became the standard cane in Fiji. In a recent letter Mr. North writes that Badila is still the favorite cane in Fiji and that, up to the present time, they have not succeeded in finding a variety that is more resistant to Fiji disease.

Control of Fiji Disease in Fiji.

The very existence of the sugar industry of Fiji was at one time gravely threatened by this disease. At the present time the industry is thriving, with the disease quite under control. The planters of Fiji hold that this has been accomplished by employing resistant varieties and by selecting only healthy canes for seed; but their system of rotation has undoubtedly been a most important factor in effecting this control. Their general practice is to take but one ratoon crop and then to keep the land under beans for a year before again planting it to cane. Early in their experience with the malady they discovered that it rarely attacks cane growing on poor soil and consequently they find it expedient to take

Figure 27.—*Fiji Disease.* Four rows of badly diseased ratoon cane. More resistant varieties are growing on either side. Photographed by Mr. North.

their seed from their poorest fields. In this way they obtain cuttings comparatively free from infection and the resulting stools are able to grow two years or more on the heaviest soils before the disease overtakes and destroys them.

While conducting entomological investigations in Fiji during the past year, Mr. C. E. Pemberton wrote on Fiji disease as follows:

> "As mentioned in a former letter, the Fiji disease is now completely under control in Fiji. I have discussed the plan of operation against this disease with many of the C. S. R. people and the independent planters. Their method has been universally the same and the results have been entirely successful everywhere. Should Fiji disease ever reach Hawaii, their successful experience in checking and almost

eradicating it will be of great value to Hawaii through the adoption of the same methods. The results have been achieved entirely through seed selection.

"The selection of seed for planting, free from outward evidences of Fiji disease, goes on as rigorously now as ever, though it is usually difficult to find stools affected by it. Specially experienced men pass along the rows and cut seed only from stools which show absolutely no signs of the disease. It is a matter of stool selection, rather than a selection of good sticks. Sometimes a vigorous stool will show one stick affected. The entire stool is left standing and goes to the mill to be ground, or, as on some estates, it is dug up and burned. This simple selection of seed from only healthy stools seems to have resulted in a complete control of the disease. I have been told by some of the independent planters that a brief laxity in such selection for a few seasons results in a quick ascendant return of the disease in all of the newly-planted fields."

Summary.

1. Fiji disease is an infectious cane disease now known to occur in New Guinea, Australia, Fiji and the Philippines.
2. The one unique symptom by which this disease may be recognized is the presence of pronounced swellings or galls in the vascular bundles of the stems and leaves.
3. A foreign body, simulating a plasmodium and occurring in each cell of the pseudo-parenchymatous gall-tissue, is held to be the parasitic agent responsible for the disease.
4. No cane variety has yet been found which is immune to Fiji disease, but a few are sufficiently resistant to permit of their profitable culture in districts where the disease is continuously epidemic, when proper control measures are practiced.
5. Fiji disease is controlled in Fiji by
   - A. the use of resistant varieties,
   - B. the use of cuttings from healthy canes only for purposes of propagation, and
   - C. the replanting of all fields after taking but one ratoon crop.

A POSSIBLE CAUSATIVE AGENT FOR THE MOSAIC DISEASE OF CORN

By L. O. Kunkel

Introduction

Because of their wide distribution, great economic importance and interest to science, the mosaic diseases of plants have been given much study during recent years. Nevertheless, the etiology of these diseases remains obscure. Some of them, as is well known, are highly infectious. This has led most students to look for a parasitic organism in the mosaic tissues. Since a causal organism has not yet been demonstrated for any mosaic disease, the assumption has frequently been made that such an organism may be present in the tissues but too small to be seen even when highly magnified. Ultramicroscopic bacteria have been suggested, but up to the present time we have no proof that such organisms exist, much less that they cause disease in plants.

During the course of some cytological and histological studies on the mosaic disease of corn, *Zea mays* L., the writer has observed that an intracellular body is always present in certain parts of diseased plants. This body is never found in the cells of healthy plants or in the healthy parts of diseased plants. The constant association of the body with the diseased condition as well as its general appearance and position in the corn cell has led to the belief that it may be a parasitic organism. The chief object of this paper is to describe the mosaic of corn and the intracellular bodies that are associated with the disease.

Distribution and Nature of the Disease

Corn mosaic is not a new disease in the Hawaiian Islands. Its presence and destructive nature were recognized by Dr. H. L. Lyon as early as 1914. In a circular letter which he sent out December 20, 1917, the following statement is made:

"An infectious chlorosis of corn has become quite prevalent in the corn fields of Hawaii during recent years. Corn is more seriously injured by this disease than is cane by yellow stripe disease. There is some reason to believe that the cane and corn diseases are identical, but this conclusion has not yet been fully demonstrated."

The first description of corn mosaic was published by Weston (9) in the 1917 Report of the Guam Agricultural Experiment Station. Weston became familiar with the disease during his visit to Hawaii and was able to identify it in the corn fields of Guam. Regarding the disease in Guam he says:

"This trouble did not appear nearly so destructive or extensive as in Hawaii, but in one field at Umatac it was causing considerable loss."

In a recent publication, Brandes (2) reports corn mosaic in Porto Rico and in the southern part of the United States. He gives a description of the disease and, from the results of an infection experiment, in which corn aphids were used, comes to the conclusion that the virus of corn mosaic is identical with that of sugar cane and sorghum mosaic.

The writer first observed the mosaic disease of corn on a homestead near Honokaa, Hawaii, in April, 1920. It was present in a field of about eight acres of an undetermined variety. While it was not causing great damage to the field as a whole, it was severe in certain spots. All plants having the disease were badly dwarfed and it was easy to see that it might become very destructive. Since this first observation the disease has frequently been seen in gardens and small fields on the Islands of Oahu, Maui and Hawaii. In small plantings of sweet corn it has occasionally been observed to cause a total loss of crop. Under conditions such as exist in Hawaii it is one of the most destructive corn diseases known to pathologists. /

Varietal Susceptibility

During the past year a number of different varieties of corn have been grown in a garden at the Forest Nursery of the Experiment Station of the Hawaiian Sugar Planters' Association in Honolulu. The mosaic disease has been prevalent in this garden during all seasons of the year. In order to test their susceptibility to mosaic a number of different varieties of corn were planted in this garden in the fall of 1920. The varieties were planted side by side in different rows. So far as known, they were all equally exposed to infection. The following varieties were grown:

Varieties planted October 14, 1920:

Cosmopolitan	sweet	corn	Crosby's Early	sweet	corn
Black Mexican	"	"	Extra Early Adams'	"	"
Country Gentleman	"	"	Queen's Golden	pop	"
Early Fordhook	"	"	White Rice	"	"
Burpee's Earliest Catawba	"	"	White Guam	field	"
Stowell's Evergreen	"	"	Yellow Guam	"	"
Golden Bantam	"	"	Cuban Red	"	"

Varieties planted October 28, 1920:

St. Charles Yellow	field	corn	Hybrid No. 1415	field	corn
St. Charles White	"	"	U. S. Selection No. 77	"	"
Commercial White	"	"	" " No. 119	"	"
Reid's Yellow Dent	"	"	" " No. 120	"	"
Boone County White	"	"	" " No. 125	"	"
Lancaster Surecrop	"	"			

The seed used in these tests was obtained from the following sources: seed of the varieties U. S. Selections No. 77, No. 119, No. 120, and No. 125 was fur-

nished by the Office of Foreign Seed and Plant Introduction of the U. S. Department of Agriculture; seed of the varieties St. Charles Yellow, St. Charles White, Commercial White, Reid's Yellow Dent, Boone County White, Lancaster Surecrop, White Guam, Yellow Guam, Cuban Red, and Hybrid No. 1415 were obtained through the kindness of Mr. H. L. Chung of the Hawaii Agricultural Experiment Station; seed of all sweet corn and pop corn varieties was purchased of W. Atlee Burpee Company of Philadelphia, Pennsylvania. Seed of the varieties White Guam, Yellow Guam, Cuban Red and Hybrid No. 1415 was grown locally; seed of all the other varieties used came from the United States and presumably from districts free from mosaic disease.

The variety listed as Hybrid No. 1415 is one which has been developed by the Hawaii Agricultural Experiment Station in Honolulu. Seed of the variety Crosby's Early failed to germinate but the seed of all other varieties gave a good stand. Approximately sixty plants of each variety were grown.

The reactions of the different varieties of corn to the mosaic disease were watched during the past winter. The disease spread to every variety grown in the garden. However, it was much more severe on some varieties than on others. On the whole, field corn was less seriously attacked than sweet corn. It is not believed that the results of one test and especially a test with such a small number of plants can be taken as final proof of resistance in any variety. However, those varieties that became badly infected are believed to be very susceptible.

The following varieties showed high percentages of infection and suffered severely from the disease: (In sweet corn) Cosmopolitan, Black Mexican, Early Fordhook, Burpee's Earliest Catawba, and Golden Bantam; (in field corn) St. Charles Yellow, Commercial White, Reid's Yellow Dent, Boone County White, and U. S. Selection No. 119.

Varieties that showed a smaller percentage of infection and appear to be somewhat resistant are: (In sweet corn) Country Gentleman, Stowell's Evergreen, and Adam's Corn; (in field corn) St. Charles White, Lancaster Surecrop, White Guam, Yellow Guam, Cuban Red, Hybrid No. 1415, and U. S. Selections No. 77, No. 120 and No. 125; (in pop corn) Queen's Golden and White Rice.

Symptoms

Mottling or striping of the leaves and dwarfing of the plant are the most striking symptoms of corn mosaic. The leaves, leaf sheaths and rind of the stalk are mottled or striped with areas of a lighter green color. In certain instances the color in the dark green areas is more intense than is common for normal healthy tissues of healthy plants. This heightens the contrast between the light and dark green areas. The shade of green in the lighter areas varies considerably in different plants and during different stages of the disease.

Even before the leaves unfold and while they are still quite white the diseased areas appear translucent and can readily be distinguished from the portions of tissue, which on exposure to light will become deep green. As the leaf unfolds in the light and becomes green the outlines of the diseased spots or stripes become more clearly marked. The lighter color of the diseased areas is due to

a failure of these portions to develop the normal green color rather than to a fading out of the green color after it has once been produced.

As the leaf ages in the light, color changes occur in both healthy and diseased portions. The lighter green areas may take on either a deeper or a lighter green color than that which they originally showed; the deeper green areas usually become lighter in color with age. In the case of badly diseased plants the green color gradually fades from the entire leaf and it becomes a light yellow. Similar changes occur in the diseased and healthy portions of the leaf sheath and rind of the stalk.

The light green areas vary in length from a small fraction of an inch to several feet. They are usually quite narrow and are distributed along the veins, giving to the leaf a beautifully striped or mottled appearance, which is very characteristic. This striping and mottling makes the disease easy to identify. There is a great variation in the sharpness of the line of demarkation between the light and dark green areas. In some specimens this line is distinct and definite while in others the deep green passes gradually into the lighter green. Portions of leaves showing typical striping are pictured in Plate 4, figures 1, 2 and 3; in Plate 6, figure C; and in Plate 7, figure B.

Figure 1 of Plate 4 shows a badly diseased leaf with elongated stripes throughout. Figure 2 shows a less severely diseased leaf. In this case the edges of the leaf are still healthy but the central portion shows the characteristic light colored stripes. Figure 1 is from the sweet corn variety, Stowell's Evergreen; figure 2 from the field corn variety, Boone County White; and figure 3 from the sweet corn variety, Early Fordhook. Figure 3 shows a pattern made up of shorter stripes. The portion of leaf shown in Figure 4 is from the variety of field corn U. S. Selection No. 120. This leaf shows typical mottling. While striping is shown by the diseased leaves of most varieties of corn, those of U. S. Selection No. 120 always show mottling. In this variety the light green areas are rather broad and not so long as in most other varieties. The patterns shown by the diseased leaves of U. S. Selection No. 120 are almost exactly like those shown by the leaves of certain sugar cane varieties when suffering from the yellow stripe disease.

So far as the writer has observed all mosaic corn plants are more or less dwarfed. This symptom is perhaps even more striking than the striping and mottling of the leaves. The degree of dwarfing varies with different varieties. In general it may be said that very susceptible varieties suffer more from dwarfing than do the more resistant ones. The age of the plant at the time it is attacked is also an important factor. Plants that become diseased while young are severely dwarfed, even in the more resistant varieties. Plants attacked before they reach the tasselling stage never produce good ears of corn. If the plant is several feet high before contracting the disease the dwarfing will be less marked. Even in cases of extreme dwarfing the disease does not seem either to hasten or delay the time at which plants mature. Diseased plants show their silk and tassel at the same time as the healthy ones. They frequently produce one or more small nubbins. The disease does not have much effect on the yield of plants that are attacked only shortly before they reach maturity. Mosaic

has not been observed to kill young plants outright. Mature diseased plants die and dry up earlier than do normal ones.

The dwarfing of diseased plants is chiefly due to a shortening of the internodes. This is plainly shown in Plate 8. Here a diseased and healthy plant of the sweet corn variety, Early Fordhook, are shown side by side. The plants have been stripped of their leaves in order that the length of the internodes may be more easily observed. The height of the plants may be judged by the two-foot ruler shown in the picture. Figure A of Plate 7 shows a diseased and healthy plant of White Rice pop corn. The diseased plant is much dwarfed and its stem is somewhat distorted. Figure C of the same plate shows a diseased and healthy plant of the field corn variety Reid's Yellow Dent. The diseased plant is much dwarfed and the leaves are bunched at the top of the plant. Figures D and E of Plate 6 show a diseased and healthy plant of the field corn variety Boone County White. These plates illustrate the dwarfing effect of the disease on different varieties. The diseased plant pictured in Plate 6 also shows bunching of the leaves at the top. This bunching of leaves is due to a failure of the upper internodes to make normal growth and is a characteristic symptom of the disease. Other symptoms less striking than those above enumerated are: excessive suckering, distortion and shrivelling of the stalk and premature dying of the lower leaves.

Fig. 1. A portion of a diseased stalk of the field corn variety Boone County White. The internodes are shortened and the stalk somewhat shriveled. The oblique surface of the cut end shows the cavities that are commonly present during the early stages of the disease.

It was soon observed that the disease is present not only in portions of the leaves, leaf sheaths and outer rind, but also deep within the tissues of the stalk. In the outer green portions of the plant, diseased tissues are readily distinguished by their light green color. Although less conspicuous the symptoms of disease in the deeper tissues are characteristic for mosaic. Diseased portions of tissue occur within the stalk, as much elongated pockets that are frequently separated from each other by healthy tissue. During early stages of the disease they may be distinguished from the surrounding healthy tissue by their water soaked appearance. These tissues later take on a slightly yellow or brown color. In a still more advanced stage all or a part of the cells in the pockets collapse and elongated cavaties are left within the stalk. A cross section of a stalk during this stage of the disease shows cavities such as are pictured in figure 1. The cut ends of diseased and healthy stalks of the variety Boone County White are shown in figures A and B of Plate 6. In severe cases the pockets become greatly enlarged and all, or almost all, of the tissues of the stem may be involved in

partial collapse. This is the condition in most of the stalks that show shrivelling and severe dwarfing.

If, as is frequently the case, the plant reaches considerable size before becoming infected, the upper internodes will show progressive stages of severity of attack. The lowest internode to show the disease may contain only one colony or pocket of diseased tissue. The next internode above will usually show a greater number of pockets, and succeeding internodes still higher up on the plant will show increasing numbers of diseased groups of cells. The disease has not been observed in the roots.

The Intracellular Bodies

The observation of mosaic plants showing such marked symptoms as those described above leads one to inquire as to what takes place within the diseased cells and tissues. It is natural to expect that such profound changes as are produced in the outward appearance of the plant will be accompanied by equally important changes within the tissues of which it is composed. It was in the hope of observing these changes and of learning, if possible, how they are brought about that a cytological and histological study of diseased plants was undertaken. On account of its large size and succulent tissues the corn plant is especially well suited for such a study.

The plants used in this work have been grown in a garden at the Forest Nursery of the Experiment Station of the Hawaiian Sugar Planters' Association. Mosaic occurs naturally in this garden and there has been no difficulty in getting plenty of diseased plants for study.

Tissues from both diseased and healthy plants were fixed in Flemming's weaker solution and in Flemming's stronger solution. This material was washed, carried through different grades of alcohol and embedded in paraffin in the usual way. It was then sectioned by means of a microtome and stained. Most of the sections were cut ten microns thick. Several stains were used including Zimmermann's stain, Flemming's triple stain, and Delafield's Haematoxylin. The best results were obtained with material fixed in Flemming's weaker solution and stained with Flemming's triple stain. It was rather difficult to obtain satisfactory preparations. This is partly due to the fact that the diseased tissues tend to stain too deeply.

Most of the cells in the diseased portions of the stalk and in the light green portions of the leaves and leaf sheaths contain one or more small bodies that may be clearly differentiated from the corn cell nucleus and other cell organs by proper methods of staining. These bodies have been studied with much interest for it is believed that they may be living organisms and may be responsible for the disease. Although the parasitic nature of the bodies has not been proved, the cells that contain them will, for the sake of convenience, be referred to as host cells.

The intracellular bodies show great variation in size. During early stages of the disease they are so minute that it is difficult to find them even in well stained preparations under high magnification. One of the small bodies closely applied to the host cell nucleus is shown in figure M of Plate 5. These tiny

bodies appear to increase in size gradually as the diseased condition of the cell becomes more and more severe. They finally reach such a size that they become conspicuous.

The bodies vary greatly in shape. They are never spherical but irregular and amoeboid, and always associated with the host cell nucleus. Their positions in the host cells are very characteristic. They are always near the nucleus and usually appear to be attached thereto. The host cell nucleus becomes enlarged. It is often deeply embedded in the body and at times appears to be engulfed. Careful observations have, however, led to the opinion that the body seldom, if ever, entirely surrounds the nucleus.

Several of the positions occupied by the bodies in the host cell may be described as typical. One of the most interesting cases is that where the body is located a short distance from the nucleus but seems to be attached to it by means of a thin, veil-like appendage. At times this structure is wrapped around the nucleus; at other times it appears as a short middle piece connecting the nucleus with the body. In figure B of Plate 5 it is shown hanging over the nucleus. Another characteristic position of the body on the nucleus is that shown in figure C of Plate 5. Here it extends out from the nucleus like a tall cap. A somewhat similar position is taken by the one shown in figure L of the Plate. While the host cell usually contains only one of the bodies, numerous cases have been observed where two or more of them are clustered around the same nucleus. In figure J, four small ones are closely applied to a nucleus. The positions on the nuclei shown in figures A, D, E, F, H, and K are less characteristic than those above described but they are positions frequently met with in any study of the relation of these bodies to the host cell nucleus.

In most instances the bodies appear to be naked and there is no indication that they are surrounded by a membrane. In other cases, however, a thin membrane is present. The body shown in figure K has a membrane. The fact that bodies in contact with each other do not fuse suggests that membranes are frequently present, even when difficult to demonstrate.

The substance of which the bodies are composed shows characteristic structure, which varies during different stages of the disease. The body pictured in figure E of Plate 5 has a coarse reticulate structure. That the structure is in most instances finely reticulate and resembles that of protoplasm will be seen by reference to figures A, C, D, F, H, J, L, and M. The structure shown in figure G distinctly marks a certain stage in the development of the body. Here elongated, dense strands, rather uniform in diameter and much branched appear as a tangled mass in a matrix of lesser density. By this structure the bodies may be easily and definitely identified even in unstained sections. The body in figure B exhibits a structure which is intermediate between that shown in figure G and the fine reticulum which is more commonly seen. The body shown in figure K is from a cell in a late stage of disease. The structure of this one is quite compact. Small granules are scattered about in the reticulum.

All attempts to demonstrate a nucleus in the bodies have failed. This has been a disappointment, for the presence of nuclei would prove them to be living organisms and would leave little doubt as to their relation to the disease. Certain

deep staining granules, which might possibly be nuclei, are frequently present. The shape of the granules in different bodies varies somewhat but they are of fairly uniform size. They tend to be angular rather than smooth and spherical, sometimes occur in pairs and occasionally are attached in pairs. Granules are shown in the bodies pictured in figures D, F, J and L of Plate 5. The fact that granules are not always present would seem to indicate that they are not nuclei. Their absence from some of the bodies is not, however, final proof against the assumption that they may be nuclei. It has frequently been observed that the plasmodia of *Plasmodiophora brassicae,* Wor. (10), do not always contain nuclei.

Vacuoles are usually though not always present in the bodies. During a certain stage in their development they often contain a single large vacuole. Figure A of Plate 5 shows such a stage. Figure H of Plate 5 shows a body that contains many small vacuoles. Other bodies containing vacuoles are shown in figures B, E, F and K.

The dark objects appearing as cell inclusions, shown in the photomicrographs on Plates 9 to 15 inclusive, are the bodies in question. In several of these photographs it is impossible to distinguish clearly the bodies from the host cell nuclei to which they are attached. The photomicrograph figure C of Plate 9 shows some of the bodies highly magnified. The body shown in the cell marked X is the same as the one shown in figure A of Plate 5.

Observations on Living Cells

Many free hand sections have been made of living stalk tissue showing different stages of disease. These were mounted in water and studied under high magnification. The microscopic study of such living material has brought to light some interesting facts. Some of the changes that take place in the cells of the diseased pockets of tissue can best be observed in living material. During early stages in the development of disease colonies, the cells of which they are composed are more turgid than those of the surrounding normal tissue. They are also unduly rich in protoplasmic content and their nuclei are from one to several times larger than the nuclei of similar normal cells.

Such cells are best described by saying that they are in a state of great protoplasmic excitation. Rapid streaming movements occur in the cytoplasmic strands, which are especially numerous. Although these cells may be located in mature tissues they resemble cells in the meristematic tissues, and always contain one or more small irregular shaped bodies either attached to or located very near the cell nucleus. These bodies are denser and more opaque than the cytoplasm of the corn cell. In favorable material they can easily be recognized after they have once been observed. The bodies are sometimes moved slightly by the vigorous streaming of the host cell cytoplasm. They have not been seen to show independent movement or to change their shape. They often contain one or more vacuoles. Their general appearance and position in the living cell is much the same as in the stained preparations. Figure I of Plate 5 was drawn from a living cell. This cell contains two nuclei, with a foreign body attached to each; one nucleus is below the plane from which the drawing was made.

Effect of the Disease on the Host Cell

Mention has already been made of the enlargement of the host cell nucleus. Nuclei of diseased cells are always much larger than those of similar normal cells. Whether or not the enlargement is due to the presence of the bodies in diseased cells is a question which can not at present be answered. The regularity with which the bodies are applied to the enlarged nuclei suggests that they are the stimulating agents. The host cell nucleus does not change its shape as it enlarges except that it often becomes flattened on that portion of its surface against which the foreign bodies are impressed. Diseased nuclei gradually lose their chromatin and are less dense than normal nuclei. The nucleolus enlarges to a degree corresponding to that of its nucleus. The nuclear membrane becomes thin. Although binucleate diseased cells are not uncommon, diseased nuclei have not been observed to divide.

The disease usually causes the host cell to enlarge. The effectiveness of the growth stimulus seems to depend on the age of the cell at the time it becomes infected. Many diseased cells die and collapse. This may happen even when little or no abnormal growth has taken place. However, cells that make considerable growth die and break down earlier than cells that respond more slowly. It is the breaking down of groups of diseased cells which, in the stalk tissues, produces the cavities that have already been described and pictured. The final collapse of the cell seems to be the result of a softening of the cell walls. Before their disintegration the walls become thickened and are frequently covered with small crystals. The crystals may occur scattered about over the walls or they may produce a fern-like structure over parts of the walls. Saprophytic fungi and bacteria are frequently numerous in badly diseased tissues. These organisms, no doubt, take part in the final breaking down of cell walls.

The Disease in the Tissues

The effects of the disease can best be observed in the inner parenchymatous tissues of the stalk. It is in these tissues also that the foreign bodies are most easily demonstrated. This is chiefly due to the fact that the cells are large and free from chloroplasts. The intracellular bodies have never been found in the cells of the growing point, but they frequently occur a short distance back of this region. They have been found in the cells of every tissue of the stalk and leaf. In most instances the tissues of the upper internodes of the stalk are more thoroughly infected than are those of the lower internodes. Although frequently found in cells of the main stem of the tassel, the bodies seldom occur in the tassel branches. They are often present in the cells of the pith of the cob but have never been found in the outer portions of the cob or in the corn grains. Roots from badly diseased plants have been carefully examined but the bodies have not been seen in the cells of root tissue. They are always present in the light green parts of leaves and leaf sheaths but are never found in the dark green portions.

Sections of stem tissue taken from moderately diseased internodes show the bodies in tissues adjacent to certain of the fibro-vascular bundles but absent from other surrounding tissues. Such a condition is seen in figures A and B

of Plate 9. The cells that contain the foreign bodies are diseased and some of them show early stages of collapse. Cells in the surrounding tissues are free from the bodies and appear healthy. From such fibro-vascular bundles the disease seems to spread gradually to other parts of the stem tissue. After the disease becomes general throughout the tissues of the stalk the distribution of the bodies bears no relation to the fibro-vascular bundles. Such tissue in an early stage of disease is shown in figure A of Plate 10. Figure B of the same plate shows a similar condition but a more advanced stage of the disease. The bodies in the cells of this section are much larger than those shown in figure A. The host cells have started to grow and their disintegration will soon begin.

Stages in the formation of the cavities, which have already been described as a symptom of the disease in the stalk, are shown in Plates 11 and 12. An early stage in the formation of a cavity near one of the disease distributing vascular bundles is pictured in figure A of Plate 11. Figure B of the same plate shows a somewhat later stage. Here the breaking down of the cells is well under way but has not gone far enough to produce a cavity. Figure A of Plate 12 shows a still later stage. A small cavity appears in this section. The size of the cavity will gradually increase with the growth and disintegration of surrounding cells. The foreign bodies in the enlarged cells bordering on the cavity are much larger than those in the cells that have not yet started to grow. This suggests that the cells around the cavity became infected earlier than those further away. Figure B of Plate 12 shows the disease in younger tissues than those pictured in Figure A. Some of the cells of this section have reacted strongly to the growth stimulus. The sections pictured in figures A and B of Plate 13 show how the growth reaction follows the development of the bodies within the cells. As the size of the bodies increases the cells that contain them show increasing signs of disease. Late stages in the breaking down of stem tissue are shown in figures B and C of Plate 14.

It is somewhat difficult to demonstrate the presence of the foreign bodies in the cells of diseased leaf tissue. They are, nevertheless, just as constantly present in these tissues as in those of the stalk. And furthermore, the changes that accompany their presence in the leaf are essentially the same as those that have already been described for the stalk. They become distributed in the tissues along certain of the veins. The cells that contain them are stimulated to abnormal growth. This causes a slight thickening of the leaf in diseased portions, as is plainly shown by the sections in figure A of Plate 14 and in figures A and B of Plate 15. In these sections the mesophyll of the normal green tissue appears almost black. It will be seen that the line separating the diseased from the healthy tissue is very sharp. The chloroplasts in the cells that contain the foreign bodies never have the deep green color that those in healthy cells possess. As the leaf matures some of the hypertrophied cells in diseased parts collapse. This may cause these portions to become thinner than the green areas. These morphological changes in the leaf result from the growth and final collapse of diseased cells. Figures C and D of Plate 15 show cross sections of leaves. These sections are highly magnified. The foreign bodies can be seen in the diseased cells. They appear as dark objects and are distributed through a small part of the section.

The cells of the tissues on either side are free from the bodies. These tissues were deep green in color while the portion that contains the bodies was light green in color. The light green portions of the leaf and the portions in which the foreign bodies are present are coextensive. Chlorosis in the leaf is definitely associated with the intracellular bodies.

The growth of the cells in diseased tissues leads to incipient gall formation. Early in the attack, diseased parts of the stem appear water soaked and are more turgid than the surrounding tissues. Likewise, diseased portions of the leaf become thickened. The growth of the infected cell is, however, very limited. Death and disintegration prevent the formation of well defined galls.

The Relation of Corn Mosaic to the Yellow Stripe Disease of Sugar Cane

It has frequently been suggested that the mosaic disease of corn and the yellow stripe disease of sugar cane are identical. They have many symptoms in common and are known to have approximately the same geographical distribution. Brandes (2) reports that he has been able to transfer the cane disease to sorghum and from sorghum to corn by means of the corn aphid, *Aphis maidis*. While there may still be some question as to the identity of the two diseases, they are undoubtedly closely related.

The mosaic pattern shown by the leaves of the field corn variety U. S. Selection No. 120 is exactly like that shown by some varieties of sugar cane. Corn mosaic frequently produces markings on the stalk similar to those produced on cane by the yellow stripe disease. Both diseases cause shrivelling of the stalk and in both cases this is due to the production of internal cavities and the collapse and drying out of necrotic tissues within the stalk. They both cause more or less dwarfing of their host plants. Both diseases are infectious and at times spread rapidly in the fields.

It must be recognized, however, that the two diseases differ in certain respects. Only in a few varieties does the cane disease approach the severity that is commonly shown by the corn disease. The cane disease has never been seen to produce the long, light green colored stripes that are so common on the leaves of diseased corn plants. Up to the present time the writer has been unable to observe intracellular bodies associated with the cane disease. Moreover the abnormal growth of cells, which is such a common symptom of the disease in corn has never been observed in the diseased tissues of cane.

Matz (7) has recently published a very interesting article in which he gives an account of some histological studies on cane tissues suffering from the yellow stripe disease. He points out that certain of the cells in diseased cane tissue are filled with a granular substance which he refers to as a plasma and suggests that this may be the plasmodium of a living organism. He says:

> "Microscopic sections of the discolored areas in yellow-striped cane stalks show that some parenchyma cells are full of a more or less hardened or compact, densely but finely granulated and slightly browned plasma."

The writer is able to confirm the observations of Matz as regards the occurrence in diseased cane tissue of cells filled with a hardened, granular, slightly

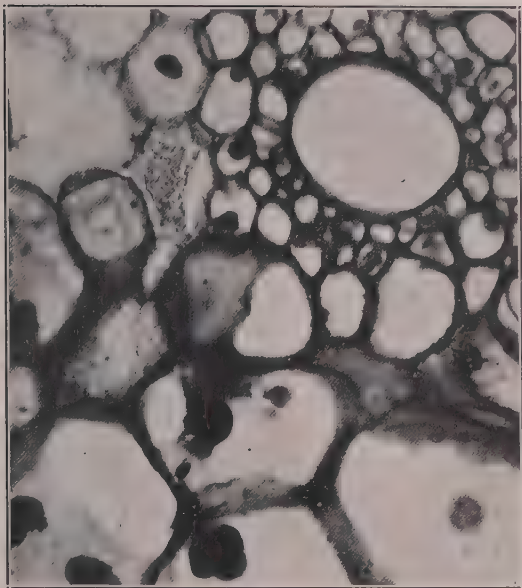

Fig. 2. A cross section of diseased corn stalk tissue, showing cells filled with a granular substance.

brownish substance. Such cells are constantly associated with the disease in mature tissues. This substance, however, does not resemble any of the plasmodia with which the writer is acquainted. It does not stain like protoplasm, does not show a protoplasmic structure, is not vacuolate, does not contain any structure that could be taken for a nucleus, and is not plastic. When crushed, this substance breaks up into irregular shaped angular masses which keep their form indefinitely and do not dissolve in water.

Cells filled with exactly the same kind of granular material are also present in the stalk tissues of corn suffering from mosaic. They are not abundant, however, and they have not been observed in corn leaves. Such cells, when present in the corn stalk, are always located near one of the small cavities or near a fibro-vascular bundle from which the disease seems to have spread. Several corn cells filled with the granular substance are shown in figure 2. What this substance may be or what relation it may have to either the cane or the corn disease the writer is unable to suggest.

Certain stages in the breaking down of diseased cane cells resemble closely some of the stages in the disintegration of corn cells. Both diseases cause thickening and softening of the cell walls. A frost-like crystalline deposit exactly like that observed on the walls of corn cells is frequently present on the walls of diseased cane cells. Necrosis in the parenchymatous tissues of the cane stalk resembles that in the corn stalk.

Other Monocotyledonous Hosts

Corn and sugar cane are not the only monocotyledonous plants that suffer

from a mosaic disease. Brandes (1) reports mosaic on sorghum, rice, millet, crabgrass, foxtail, and *Panicum dichotomiflorum*. It seems to be a common disease on narcissi, hyacinths and tulips in the bulb-growing regions of Holland where it is referred to as the "gray disease" (4). During a recent visit to Hawaii, Dr. R. J. Tillyard, chief of the Biological Department of the Cawthron Institute of Science and Research at Nelson, New Zealand, informed the writer that the growers of New Zealand flax, *Phormium tenax* Forst., are troubled with what is known to them as "yellow leaf disease." From his description it seems probable that the disease in question is mosaic.

Mosaic has been observed on several different monocotyledonous plants in Hawaii. Honohono grass, *Commelina nudiflora* L., is one of the most common weeds in the sugar cane fields. Many of these plants are attacked by a mosaic disease. What appears to be mosaic has several times been observed on the Chinese banana, *Musa Cavendishii* Past., and on bamboo, *Bambusa vulgaris* Wendl. A disease closely resembling the yellow stripe disease of sugar cane occurs on *Dianella odorata* Bl. Mosaic has frequently been observed on Canna, *Canna Indica* L., and on *Hippeastrum* sp.

Discussion

In any consideration of the significance of the intracellular bodies associated with corn mosaic two questions naturally arise: (1) Are the bodies living organisms? And (2) if they are organisms, do they cause the disease? While it is not possible at the present time to prove that the bodies are living parasites, there is considerable evidence in favor of this view. They grow, show a structure like that of protoplasm, stain like protoplasm, and tend to be amoeboid in shape. It seems hardly probable that waste products accumulating in diseased cells or the products of protoplasmic degeneration would show these characteristics and would always be so closely associated with the host cell nucleus. Some well known intracellular parasites occupy exactly the same position in the cells of their respective hosts that the bodies of corn mosaic occupy in the cells of the corn plant. Orton and Kern (8) have called attention to the fact that the swarm spores of *Chrysophlyctis endobiotica* Schilb. ". . . . show a very marked tendency to cluster around the nucleus of the host cell." The plasmodia of *Spongospora subterranea* (Wallroth) Johnson show the same tendency. During the later stages of their life within the cells they are always closely applied to the host cell nucleus (5).

If the bodies are living organisms there would seem to be little question but that they are responsible for the disease. They are always present in the cells of diseased plants but are never found in the cells of healthy plants. No other foreign organism has been found in the diseased tissues. Moreover, the bodies are present in such numbers and are distributed in such a way as to account for the disease. If they could be grown on media in pure culture, and when inoculated from such cultures into healthy plants, would produce the typical mosaic disease and if from the plant in which the disease had been artificially produced the bodies could be recovered and again grown in pure culture, we would have unquestionable proof that they cause the disease. It must be remembered, how-

ever, that up to the present time very few intracellular parasites have been grown *in vitro* and it is hardly to be expected that the much desired cultures will be easy to obtain.

The bodies of corn mosaic show very little resemblance to the plasmodia of *Plasmodiophora brassicae* Wor. or to any of the other parasites belonging to the Plasmodiophoraceae. They are also unlike the deep staining foreign bodies constantly present in the cells of the galls of the Fiji disease of sugar cane (6). They call to mind the intracellular bodies associated with certain of the virus diseases of animals. The well known Negri bodies (*Neuroryctes hydrophobiae*) are always present in the brain cells of dogs and other animals suffering from rabies. Other bodies (*Cytoryctes variolae*) occur with equal constancy in the skin cells of man and apes infected with smallpox. None of these bodies have yet been grown in pure culture and final proof that they are etiologically related to the diseases in question has not been given. It has not even been proved that the bodies are living. Nevertheless, there is much morphological evidence that they are organisms and Calkins (3) has expressed the opinion that they are protozoan in nature.

Summary

(1) A foreign body believed to be a living organism is invariably present in diseased cells of mosaic corn plants.

(2) The body is irregular in shape and always occupies a position on or near the host cell nucleus. It usually shows a definite reticulate structure, stains like protoplasm and is frequently vacuolate.

(3) The distribution of the intracellular bodies corresponds exactly with the distribution of the light green color in diseased leaf tissue. In the stalk, they are present in the cells of diseased tissues but absent from the cells of healthy tissues.

(4) It is suggested that the bodies of corn mosaic may be similar to those associated with certain virus diseases of man and animals.

(5) Corn mosaic is similar to, if not identical with, the yellow stripe disease of sugar cane.

(6) Nine varieties of sweet corn, two varieties of pop corn, and fourteen varieties of field corn have been shown to be susceptible to the disease. Several varieties are somewhat resistant but no variety is known to be immune.

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MORPHOLOGICAL STUDIES OF THE PYTHIUM-LIKE FUNGI ASSOCIATED WITH ROOT ROT IN HAWAII.

By C. W. Carpenter

Previous papers by the writer[1] have recorded the presence of a fungus of the *Pythium* type associated with root rot diseases of cane (Lahaina disease), pineapples (wilt), taro, rice, and bananas in Hawaii. Critical studies of the several strains isolated from the crops mentioned are being carried on with the object of determining the morphology of these organisms. Such studies, it is thought, may result in a better understanding of the nature of the diseases, the *modus operandi* of the causal agents and the relation of the environment as a limiting factor.

The pure culture of the fungus from cane, selected for the morphological studies chiefly to be discussed below, is the strain with which positive inoculations of cane and pineapple in pot culture have been obtained and previously reported. Other strains from cane, pineapple, and rice have been recently observed to have the same characters in general as the particular strain cited, while the strain from taro already reported as being parasitic differs in that it is a conidium-producing *Pythium*.

The cane fungus agrees very closely in both the asexual and the sexual stages with the sugar beet root rot fungus *Rheosporangium aphanodermatus* first described by Edson.[2] Such a fungus, which in a single trial was not found to be parasitic, is recorded in the writer's most recent paper.[3] Several additional strains of this sort of fungus, besides the one particularly discussed herein, have been isolated from cane and pineapples.

Edson notes the resemblance of his fungus to *Pythium* in the following language:

> "In the general character of the disease produced in seedlings and in its appearance in culture, the organism resembles *Pythium debaryanum* so closely as to be readily confused with it, except in the asexual fruiting stage.
>
> "The disease which it produces on the sugar beet is very similar to that caused by *P. debaryanum*. The fungus is even more aggressive as a parasite than *Pythium*. . . . . . . A disease of the side roots of growing beets was encountered during the course of the studies in soils which had been inoculated with artificial cultures of the fungus several

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[1] Carpenter, C. W., Hawaii Agric. Exper. Sta. Annual Report, 1918, p. 43 (rice root rot), p. 44 (taro rot). Report 1919, p. 50.
Preliminary report on root rot in Hawaii. Hawaii Agric. Exper. Sta. Press Bul. 54.
Pythium in relation to Lahaina disease and pineapple wilt. *In* Hawaiian Planters' Record Vol. XXIII, p. 142–174, 1920.

[2] Edson, H. A., *Rheosporangium aphanodermatus*, a new genus and species of fungus parasitic on sugar beets and radishes. *In* Jour. Agr. Res. Vol. IV, No. 4, 1915, p. 279–291. 5 Pls. See also Vol. IV, No. 2, p. 161–163.

[3] Hawaiian Planters' Record, Vol. XXIII, p. 167, 1920.

> months earlier. The fungus was readily isolated from diseased side roots of this beet and there appears to be no reason to doubt its causal relation to the trouble.
>
> "The causal relation of the organism to the radish disease[1] as well as to damping-off of sugar beet seedlings was confirmed repeatedly by inoculation experiments."

That the cane fungus should be identical with the sugar beet fungus is rather improbable in view of the lack of close botanical relationship of the hosts. Similarly it would be rather surprising if the several strains from such botanically diverse crops as cane, pineapples, rice and bananas should be identical. A description of the parasitic strain from cane and a comparison with *Rheosporangium aphanodermatus* follows. Some figures (Pl. 23, figs. 7–10) of the taro rot organism previously reported as parasitic are also given by way of illustrating the reduced diplanetism in *Pythium*. Studies of these Phycomycetes associated with the root rot problem will be continued.

The Cane Pythium in Pure Culture.

The cane fungus grows well in pure culture on several media, with an abundant white, fluffy, aerial mycelium in evidence after a few days. In petri dish cultures the growth is for the first few days restricted to the surface film, but it produces a copious white aerial mycelium after becoming established on the substratum. In slant cultures the mycelium grows up in the tube often filling the bore for some distance above the top of the slant. After some weeks it settles back as a gelatinous, tough layer on the medium.

Media found satisfactory for the isolation and culture of this fungus are as follows: For isolation, an agar medium more dilute than standard; bouillon 1 part, distilled water 4 parts, plus 1% dextrose and 2% agar. An oat agar similar to Clinton's, made from rolled oats, is satisfactory for the development of the sexual spores. For studies of asexual sporulation Lahaina cane cuttings were disinfected in mercury bichloride 1-1000 for four minutes, rinsed in sterile water and allowed to produce roots in sterile water. Such young roots (about 1 inch long) treated with bichloride for 2 minutes, rinsed, and placed in sterile water in petri dishes, when inoculated furnished good material for the microscopic studies of the asexual stage.

Young hyphae are 2.8 to 7.0 μ in diameter, non-septate; in plate cultures they often show a peculiar twisting of the short branches, which gives a knob-like appearance under the microscope (Pl. 16, fig. 1); the hyphae are septate in age and in fructification.

Sexual Stage.

On oat agar oogonia develop in 3 to 10 days and are rather characteristically entangled in the antheridia of which there may be several with their supporting branches surrounding the oogonium. The oogonia (Pl. 19, fig. 2; Pl. 23, figs. 1–6) are terminal, 24.0–35.0 μ in diameter, the average of 50 being 29.0 μ.

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[1] Black root disease.

Antherida are often from the same branch as the oogonium. The oospores are round, smooth, thick walled, slightly brownish in maturity, 21.0–28.0 μ in diameter, the average of 50 being 24.0 μ. In cane roots from the field the vestiges of the oogonium are 20.0–26.0 μ in diameter while the oospores are 18.0–24.0 μ, averaging 20.5 μ in diameter. In cane roots from plants inoculated with pure culture the oogonia measure 21.0–30.0 μ, and the oospores 20.0–25.0 μ, with an average of 22.0 μ.

Asexual Stage.

In water cultures the fungus is found to have invaded and occupied the cells of the young cane roots in a few days, the appearance (Pls. 20–21) being exactly similar to what we frequently find in field material.

Irregular globular or swollen bodies (presporangia), walled off at the tips of the hyphae, occur on agar plates (Pl. 18, fig. 1) rather infrequently, but in water culture they are formed in abundance (Pl. 20, fig. 1). They occur in the water near the surface of the root, and similar swollen bodies also fill numbers of adjacent root cells (Pl. 21, fig. 1). Resting spores are likewise formed abundantly in the cells of the cane root in water cultures.

The presporangia (Pl. 18, fig. 1; Pl. 22, figs. 1–4) vary greatly in size and shape, but they generally consist of several closely attached spherical masses, densely and finely granular and slightly brownish in color. The portions are characteristically more globose than represented by Edson for *Rheosporangium aphanodermatus*. If the water is frequently changed by means of a sterile pipette, some of the presporangia can be induced to develop in a few hours (Pls. 18–19). The contents of the presporangia flow out through a long, narrow, tubular process,[1] forming a delicate, spherical body in which the zoospores are differentiated. This is the true sporangium, if we follow Edson. The presporangia, when emptied, are scarcely to be seen, so thin are their walls, and so completely are their contents drawn out to form the sporangium.

The sporangia are spherical, dense, protoplasmic masses varying greatly in size. Their size seems to be determined by the size of the presporangial mass. As cleavage begins, the thin, hyalin, scarcely visible wall of the sporangium may be seen surrounding the contents at a distance of about 15.0 μ.

Streaming of the presporangium to form the sporangium in one case occupied eight minutes; a rocking movement began four minutes later, with cleavage lines well defined twelve minutes subsequently. Individual movement of the zoospores began in another two minutes. The zoospores were liberated in eight minutes, or thirty-four minutes from the formation of the sporangium. Another case similar to many which have been seen is sketched in Plate 22.

Of ten sporangia the average diameter of the contents was 48.0 μ and of the hyalin sporangium, 65.0 μ. The number of zoospores varies from four to over a hundred estimated; generally in culture the number was eight to twenty-four estimated; the average size of zoospores being 10.0–12.5 x 7.5–

[1] Considerably longer proportionately than shown in Pl. 22, fig. 5.

$9.5\ \mu$. Two cilia are attached at the sinus. After rounding up, the zoospores have been observed to germinate in the course of an hour, with a single germ tube. Under suitable conditions, this grows out into a hypha capable, conceivably, of attacking roots.

The fungous hyphae extend themselves through the cells of the young root, apparently being able to enter the root and penetrate the cell walls without difficulty. Although the fungus can at times be seen to grow intercellularly, it is generally to be found within the cells. After a short time (a few days) the mycelium has appropriated the cell contents more or less completely and to a similar degree occupied the interior of the cell, often being tightly packed therein. This is accompanied by the development of the hyphae into swollen, globular, thin walled protoplasmic masses, which the writer considers to be presporangia.

The resting spores are formed generally later than the zoospores in artificial cultures, and this appears to be the general rule in the roots, though the conditions of the environment rather than the mere duration of time determine the sequence.

Judging from observations of this cane fungus in the roots and in culture, the life cycle of the fungus in the field may be outlined by analogy, as follows:

In a cane root in wet soil, after the mycelium has developed as above outlined, zoospore formation is the rule, the mycelium emptying into the presporangia, which in turn empty into the sporangia, and scatter as zoospores. It is considered probable that the tube of delivery of the presporangium is capable of penetrating the cell walls, thus liberating the zoospores in the water outside the root. The empty mycelium and inconspicuous, empty presporangia remain as evidence of the cause of the disease. No traces of the sporangium are to be seen after the liberation of the zoospores. Under less favorable conditions (drier soil?), the fungus forms the relatively conspicuous oospores in the root cells. It is thought that if this is indeed the train of developments as they occur in the field, our frequent inability to find the fungus present in sufficient abundance to account for the damage to the root system of cane and pineapples is in considerable measure explained.

Pythiaceae and the Genus Rheosporangium Edson.

Edson considered his sugar beet fungus to belong in the *Saprolegniaceae*. He used the classification of Minden,[1] and finding no appropriate genus of the *Saprolegniaceae* to accommodate his fungus, established a new genus, *Rheosporangium*. Although the present writer has not been able to consult Minden's classification, he doubts the wisdom of this course for the reasons given below. It seems more natural to include this fungus under the genus *Pythium* of the *Pythiaceae*, whether we consider the latter as allied more closely to the *Saprolegniaceae* or to the *Peronosporaceae*.

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[1] Minden, M. D. von. Pilze. *In* Kryptogamenflora der Mark Brandenburg, Bd. 5. Heft 3–4, 1911–12.

For a clear presentation of Edson's point of view the author quotes the following paragraph:[1]

> "The fungus under consideration seems to present a third and hitherto undescribed type of diplanetism, in which the first mobile period consists in the migration of the entire uncleaved sporangium and its contents from the presporangium. This type of egress is new. In all related forms previously described the spores are differentiated before migration. This distinction seems sufficiently important to justify its recognition as of generic rank. The uncleaved protoplasm rather than the differentiated spore migrates."

It seems to the writer that the type of reduced diplanetism Edson describes is essentially what we have throughout the group *Pythium*.[2] In *Pythium*, according to Butler's[3] description of *P. debaryanum*, we have a sporangium which might well be called a presporangium since the contents flow out undifferentiated through a beak, the differentiation taking place in a thin walled vesicle. (Cf. Pl. 23, fig. 10.) This primary migration is not essentially different from that in *Rheosporangium*. The wall of this vesicle in the latter genus is the wall of the protoplast which is delivered and draws out with it the contents of the sporangium. Butler[4] gives the origin of this vesicle in *Pythium* as the thin membrane at the tip of the beak, for he says (p. 34): . . . . . . "the beak blows up into its vesicle." (p. 28): "The beak itself is a protrusion of the endosporal lining of the sporangial wall." Butler made a general survey of the group, and considered the phylogenetic relationship therein. He refers several times to the migration of the uncleaved sporangium. In speaking of the relation of *Pythium* to the *Saprolegniaceae*, he says (p. 50):

> "As far as the thallus is concerned, and in certain of the cruder details of reproduction the aquatic species of *Pythium* approach *Saprolegniaceae*, particularly *Pythiopsis* and to a lesser degree *Aphanomyces*. But these resemblances are superficial and are probably partly connected with the environment. In the essential points in which affinities must be looked for, the divergence is considerable. *Thus the zoospores are never liberated in a bladder as always occurs in Pythium.*[5]

And further on, in speaking of the affinity between *Pythium* and the *Peronosporaceae:*

> "But the genus *Pythium* is separated from all the rest by liberating its zoospores in an imperfectly differentiated state into a bladder at the mouth of the sporange, in which differentiation is completed."

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1 Jour. Agr. Res. Vol. IV, No. 4, p. 290, 1915.

2 See Engler, A., and Prantl, K. Die Naturlichen Pflanzenfamilien. Teil I. Abt. I, p. 104.

3 Butler, E. J. *Pythium debaryanum* Hesse. *In* Memoirs of the Dept. of Agr. India, Vol. V, p. 262–266, 1913.

4 Butler, E. J. An account of the genus Pythium and some Chytridiaceae. *In* Memoirs Dept. Agr. India, Vol. I, No. 5, p. 1–158, 1907.

5 Italics, the writer's.

The fungus agrees rather closely with the species of *Pythium* which Butler[1] has described and provisionally called *P. gracile* Schenk. Subramaniam[2] has recently described such a fungus which differs from Butler's *P. gracile* in not being an algal parasite. He says in part:

> "The mycelium is composed of much branched hyphae, sometimes showing false dichotomy, very variable in breadth, from 3.0 to 8.0 μ. Septation occurs very irregularly in old cultures. *Irregular swellings on the hyphae were quite common*[3] when grown in plant tissues or in water cultures. . . . . . . *The irregular swellings on the hyphae serve as reservoirs of protoplasm and under favorable conditions sporangia may develop from them. It is not uncommon to find empty cells at the base of the sporangial hyphae.*[3]

Subramaniam considers this a new species and names it *P. Butleri*. The close similarity of this species to the cane fungus is best shown by quoting his description *verbatim:*

> "*Pythium Butleri n. sp.* (Subramaniam).
>
> "Mycelium composed of much branched hyphae, sometimes showing false dichotomy, the main strands being 3 to 8 μ broad and the lateral ramifications thinner. Irregular swellings quite common on the mycelium, which is septate in old stages. Sporangia lateral, elongated, slightly swollen at the tip. Zoospores few to 35 in number, bean-shaped, bi-ciliate, measuring when moving 8 to 12 μ, 6 to 8 μ in diameter, and after coming to rest 7 to 11 μ. Oogonia lateral or intercalar, spherical or subspherical, thin walled, and measuring 18 to 33 μ (average 26 μ). Antheridia terminal or intercalar (when they are usually on a different hypha from that bearing the oogonium), or hypogynal (when they are on the same hypha as the oogonium), knob-shaped. Oospores round, smooth, hyaline or light yellowish when fully mature, thick-walled, never filling the oogonium completely, 13.5 to 25.3 μ in diameter (average 21 μ). Oospores germinate by a germ tube, not by zoospores. Parasitic on *Nicotiana Tabacum, Zingiber officinale, Carica Papaya, Capsicum annuum,* and capable of attacking, when artificially inoculated, *Solanum tuberosum* and *Ricinus communis.*"

Some data on the fungi *Rheosporangium aphanodermatus* Edson, *Pythium Butleri* Subramaniam and the cane *Pythium* are tabulated for convenience of comparison. The writer considers the cane fungus morphologically the same as *R. aphanodermatus* and *P. Butleri.*

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[1] Butler, E. J. *In* Mem. Dept. Agr. India, Vol. I, No. 5, p. 67-71. 1907.

[2] Subramaniam, L. S. A Pythium disease of ginger, tobacco, and papaya. *In* Mem. Dept. Agr. India, Vol. X, No. 4, p. 181–194 (6 Pls.), 1919.

[3] Italics, the writer's.

TABLE I

Comparison of certain characters of *Rheosporangium aphanodermatus*, *Pythium Butleri*, and the cane *Pythium*.

	Young Hyphae	Zoospores	Oogonia		Oospores	
			Limits	Average	Limits	Average
R. aphanodermatus ..	2.8–7.3 μ	12.0x7.5 μ	22.0–27.0 μ		17.0–19.0 μ	
P. Butleri	3.0–8.0 μ	8.0–12.0x6.0–8.0 μ	18.0–33.0 μ	26.0 μ	13.5–25.3 μ	21.0 μ
Cane *Pythium*	2.8–7.0 μ	10.0–12.0x7.5–9.5 μ	24.0–35.0 μ (Culture)	29.0 μ	21.0–28.0 μ	24.0 μ
			20.0–26.0 μ (Cane root from field)		18.0–24.0 μ	20.5 μ
			21.0–30.0 μ (Cane root in water culture)		20.0–25.0 μ	22.0 μ

SUMMARY.

1. The Pythium-like fungus previously reported as an active factor in the root rot disease of cane (Lahaina disease) is morphologically identical with *Rheosporangium aphanodermatus* Edson and *Pythium Butleri* Subramaniam.

2. The Pythium-like fungus previously reported as associated with the root rot of pineapples (wilt) and rice is similar in its morphology to the cane Pythium.

3. A taro rot fungus previously reported as like Pythium has been found to be a conidium-producing Pythium.

4. The writer considers that the cane fungus manifests a type of diplanetism in the asexual stage allied to that in the conidium-producing Pythiums, and prefers to classify it in the genus *Pythium* rather than in Edson's new genus *Rheosporangium*.

A CONTRIBUTION TO A CHECK-LIST OF SUGAR CANE FUNGI

Compiled from the Literature by

Edw. L. Caum

The following is the result of an attempt to list all the fungi which have been reported as occurring on the sugar cane *(Saccharum officinarum* L.), either as parasites or saprophytes, in any part of the world. It is necessarily incomplete, but will serve as a preliminary list.

Parasites, or fungi accredited with parasitic tendencies, are listed in **bold-face,** saprophytes in ordinary type, and synonyms and common names in *italics*. To this check-list is appended a brief classification of the fungi, following Clements and Saccardo, and a bibliography explaining the abbreviations used in citing names of publications.

The following explanation may be in order. The fungi are listed alphabetically by genera, the species being in the same order under the genus. The name of the author is given, followed by the common name of the fungus, if there is one. The next line gives, in abbreviated form, the name of the periodical in which the description was originally published, followed by the volume number. page and date of publication. In case of a change in nomenclature, the secondary description is cited in the same form. An asterisk (*) preceding either or both of these lines indicates that the reference has been verified by comparison with the publication indicated. A figure enclosed in parentheses, preceding the volume number, indicates the series, in the case of a periodical, or the edition, in the case of a text-book. In the case of pamphlets or single volumes, no volume number is given, the page number being indicated by 'p'. In references to certain periodicals where the parts were paged separately, the number of the part referred to is enclosed in parentheses and placed between the volume and page numbers. Following the citation is a short note relative to the occurrence of the fungus.

In some cases it has not been possible to obtain all this data, and the place assigned to it is therefore left vacant. The same is true in the list of publications, where the places or dates of publication could not be ascertained.

A number of the fungi listed, particularly the saprophytic forms, undoubtedly occur in sugar-growing countries other than those named in the list, but they have not been mentioned in the literature as occurring on sugar cane in those places, as far as could be ascertained.

It is probable that, as further information is gathered, additions and corrections to this check-list will be published.

A

Acremoniella atra (Cda.) Sacc.
*Corda (Acremonium), Ic. fung., 1:11. 1837.
*Saccardo (Acremoniella), Syll. fung., 4:302. 1886.
Corda gives the habitat as 'in foliis putridis Pandani', while Saccardo lists it as occurring as a saprophyte on the stem and leaf of the sugar cane.

Acremonium atrum Cda. See Acremoniella atra (Cda.) Sacc.

Acrostalagmus albus Pr.
*Preuss, Linnaea, 24:126. 1851.
Preuss gives the habitat as 'in ligno Alni glutinosae et in foliis Citri limonum', while Saccardo lists it as a saprophyte on the leaf and stem of the sugar cane.

Acrostalagmus albus Pr. var. dichotoma Speg.
*Preuss, Linnaea, 24:126. 1851.
Spegazzini, An. Mus. Nac. B. A., (2) 3:333. 1899.
Reported on piles of rotting cane leaves in Argentina.

Acrostalagmus cinnabarinus Cda.
*Corda, Ic. fung., 2:15. 1838.
Occurs on rotting vegetation generally. Corda gives the habitat as 'auf faulenden Kartoffeln'. Saccardo lists it as occurring on sugar cane.

Acrothecium lunatum Wakk.
*Wakker, Ziekt. Suik., p. 196. 1898.
A saprophyte on cane leaves in Java and Hawaii.

Agaricus crinitus Linn. See Lentinus crinitus (Linn.) Fr.

Agaricus synodicus Kze. See Marasmius synodicus (Kze.) Fr.

Allantospora radicicola Wakk.
*Wakker, Archiev, 4:892. 1896.
Parasitic on young roots in Java and Hawaii. Also a general cane saprophyte in Hawaii.

Alternaria tenuis Nees
Nees von Esenbeck, Syst. P. u. S., 2:72. 1817.
Occurs on dead vegetation in general. Saccardo mentions sugar cane stems in particular.

Anthostomella paraguayensis Speg. See Rosellinia paraguayensis Speg.

Apiospora camptospora Penz. & Sacc.
*Penzig & Saccardo, Malpighia, 11:389. 1897.
Reported saprophytic on dead cane leaves in Java and the Philippines.

Apiosphaeria Bergii Speg.
Spegazzini, Rev. Agr. Vet., :240. 1896.
Attacks young leaves in the spindle, but not fully opened leaves, in Argentina.

Arcyria cinerea (Bull.) Schum.
Bulliard (Trichia), Herb. Fr., t.477. 1789.
*Schumacher (Arcyria), En. pl. Saell., p. 213. 1803.
Reported as occurring on dead cane leaves in Porto Rico.

Arcyria denudata Fr.
Fries, Acta R. Soc. Sci. Upsala, (3) 1:135. 1851.
On dead cane leaves in Porto Rico.

Arthrinium saccharicola Stevn.
*Stevenson, Jour. Dep. Agr. P. R., 1:223. 1917.
On dead cane sticks in Porto Rico.

Arthrobotrys superba Cda.
Corda, Prachtflora No. 43. 1839.
Occurs on dead and dying cane in Porto Rico.

Aspergillus argentinus Speg.
Spegazzini, Rev. Agr. Vet., :245. 1896.
Occurs in Argentina on green cane leaves which have been cut and bundled.

Aspergillus candidus Lk.
*Link, in Sp. plant., (4), Fungi, 6:(1):65. 1824.
Saccardo lists it as a saprophyte on cane.

Aspergillus flavus Lk.
Link, Mag. Ges. nat. Freunde, 3:14. 1809.
Occurs in Porto Rico on dead cane, especially on dead seed pieces in the ground and on material in the laboratory. Not morphologically distinct from the species which attacks the mealy-bug, Pseudococcus Sacchari.

Aspergillus niger v. Tiegh. See Sterigmatocystis nigra v. Tiegh.

Aspergillus penicillioides Speg.
Spegazzini, Rev. Agr. Vet., :246. 1896.
Grows on rotted spindles in Argentina.

Asterostroma albido-carneum (Schwein.) Mass.
von Schweiniz (Thelephora), Tr. Am. Phil. Soc., N. S. 4: 1834.
*Massee (Asterostroma), Jour. Linn. Soc., 25:155. 1889.
Parasitic (?) on cane roots in Porto Rico.

Asterostroma cervicolor (B. & C.) Mass.
Berkeley & Curtis (Corticium), Grevillea, 1:179. 1872.
*Massee (Asterostroma), Jour. Linn. Soc., 25:155. 1889.
On dead sheaths at the base of living sticks, and on trash, in Porto Rico. Stevenson considers A. albido-carneum to be a synonym.

Asterula concentrica Cke.
Cooke, Grevillea, 14:13. 1885.
Saprophytic on cane stems in India.

B

Bacillus fluorescens liquefaciens Flüg. See Bacterium fluorescens liquefaciens (Flüg.) L. & N.

Bacillus glagae Janse
Janse, Mededeel. Plant., 9: 1891.
Said by Janse to be a contributory cause of Sereh in Java, but is probably a normal inhabitant of the cane.

Bacillus pseudarabinus R. G. Sm. *Red String*
*R. G. Smith, Proc. Linn. Soc. N. S. W., 29:453. 1904.
A gum-forming bacillus from Australia.

Bacillus saccharalis Owen
*Owen, Jour. Bact., 1:239. 1916.
In cavities made by borers (Diatraea saccharalis), in Louisiana.

Bacillus Sacchari Janse
Janse, Mededeel. Plant., 9: 1891.
Said by Janse to be a contributory cause of Sereh in Java, but is probably a normal inhabitant of the cane. According to Went it is probably B. subtilis.

Bacillus subtilis (Ehrbg.) Cohn
Ehrenberg (Vibrio), Infusionsth., p.80. 1838.
*Cohn (Bacillus), Beitr. Biol., 1: (2):175. 1875.
Mentioned in connection with Pokkah-bong in Java.

Bacillus vascularum Cobb. See Pseudomonas vascularum (Cobb) E. F. Sm.

Bacterium fluorescens liquefaciens (Flüg.) L. & N.
Flügge (Bacillus), Microorg., 2:289. 1896.
*Lehmann & Neumann (Bacterium), Atl. Prin. Bact., 2:285. 1901.
Probably saprophytic. Found in cane attacked by Bacillus pseudarabinus.

Bacterium Sacchari R. G. Sm.
*R. G. Smith, Trans. Linn. Soc. N. S. W., 27:142. 1902.
Parasitic in New South Wales. Smith considers it as practically a normal inhabitant of the cane.

Bacterium vascularum (Cobb) R. G. Sm. See Pseudomonas vascularum (Cobb) E. F. Sm.

Bakerophoma Sacchari Died.

*Diedicke, Ann. Myc., 14:63. 1916.

Attacks the base of the blade and the top of the sheath. Peculiar to the Philippines.

Basisporium gallarum Moll.

*Molliard, Bull. Soc. Myc. France, 18:170. 1902.

A saprophyte on living cane leaves. Reported from Hawaii and Porto Rico.

Boletus sanguineus Linn. See Polystictus sanguineus (Linn.) Mey.

Botryodiplodia Theobromae Pat. See Lasiodiplodia Theobromae (Pat.) Griff. & Maubl.

Botrytis sp.

An undetermined species reported on dead cane leaves in Porto Rico.

C

Calonectria gigaspora Mass.

*Massee, Kew Bull., :257. 1906.

In channels made by the cane borer (Diatraea sp.) in Trinidad, B. W. I.

Cantharellus spathulatus Jungh. See Guepinia spathulatus Jungh.

Capnodium sp. *Sooty Mould*

Reported from Brazil (Bol. Agric., 17:936. 1916) as parasitic on the culm and leaves. It is probably a saprophyte. Various species of Capnodium follow infestation by mealy-bugs (Pseudococcus spp.) or leaf-hoppers (Perkinsiella saccharicida). They are probably of universal distribution.

Catenularia echinata Wakk.

*Wakker, Ziekt. Suik., p. 196. 1898.

Found in dead cane sticks in Java. This may be a form of Sphaeronema adiposum Butl.

Cephalosporium Sacchari Butl. *Wilt*

*Butler, Mem. Dep. Agr. India, 6:185. 1913.

Parasitic in cane stems in India and South Africa, and maybe in the Barbados and the United States.

Cercospora acerosum Dick. & Hein

*Dickhoff & Hein, Archiev, 8:1013. 1901.

Parasitic at the juncture of the sheath and blade. Reported from Java and the Philippines.

Cercospora Köpkei Krüg.

*Krüger, Ber. Zuck., 1:115. 1890.

Parasitic on the leaves of various species of Saccharum, including S. officinarum, in Java, Reunion, Argentina, and the Philippines.

Cercospora longipes Butl. *Brown leaf-spot*

*Butler, Mem. Dep. Agr. India, 1:(3):44, 1906.

Parasitic on cane leaves in India, Porto Rico and Trinidad.

Cercospora Sacchari v. Breda. *Eye-spot*

van Breda de Haan, Mededeel. Suiker. 1892.

(Description reprinted in the Bijl Archiev, p. 94, 1893).

Parasitic on cane leaves in Java, Reunion, Hawaii and the Philippines.

Cercospora vaginae Krüg.

*Krüger, Ber. Zuck., 2:249. 1896.

Parasitic on the sheaths in Java, Reunion, the British West Indies, Porto Rico, Santo Domingo, and probably Hawaii.

Chaetomella Sacchari Delacr.

*Delacroix, Bull. Soc. Myc. France, 13:123. 1897.

Occurs on dry cane sticks in Reunion.

Chaetomium viride Lév.

Léveillé, Ann. Sc. Nat., (3) 3:65. 1845.

Habitat given in description as on dead grasses in Paraguay. Saccardo mentions sugar cane as host.

Chaetophoma Maydis Speg.
Spegazzini,
On fallen and rotting leaves of corn, and rarely on those of cane, in Brazil, according to Saccardo, who gives the reference as An. Soc. Cient. Arg., 9:143. 1880. This is incorrect, but the proper reference cannot be located.

Chaetostroma Sacchari Mass.
Massee, Grevillea, 22:67. 1893.
On drooping cane leaves in the Barbados.

Chaetostroma Sacchari Speg. (non Mass.) See Chaetostroma saccharicolum Sacc. & Syd.
Spegazzini's description is given in the Rev. Agr. Vet., :225. 1896.

Chaetostroma saccharicolum Sacc. & Syd.
*Saccardo & Sydow, Syll. fung., 14:1130. 1899.
On rotting leaves and leaf sheaths in Argentina.

Chondromyces Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :252. 1896.
On rotting cane in Argentina.

Chromocrea gelatinosa (Tode) Seav.
Tode (Hypocrea), in Fries, Summa veg. Scand., p. 383. 1849
*Seaver (Chromocrea), Mycologia, 2:58. 1910.
On dead and dying sheaths in Porto Rico.

Chromocreopsis striispora Stevn.
*Stevenson, Jour. Dep. Agr. P. R., 1:213. 1917.
On dead cane sticks in Porto Rico.

Chytridiaceous fungus.
*Lyon, Hawaiian Pl. Rec., 22:2-8. 1919.
An undetermined form found apparently parasitic in the roots of cane affected with root-rot or Lahaina disease.

Cladoderris dendritica Pers.
On dead cane stalks in Porto Rico. Description cannot be located.

Cladosporium graminum Lk.
Link, in Sp. plant., Fungi, 1:42. 1824.
Parasitic in the interior of the stick, in Brazil.

Cladosporium herbarum (Pers.) Lk.
*Persoon (Dematium), Syn. meth. fung., p. 699. 1801.
Link (Cladosporium), Mag. Ges. nat. Freunde, 7:37. 1816.
On cane trash in Porto Rico.

Cladosporium javanicum Wakk.
*Wakker, Archiev, 4:889. 1896.
Saprophytic on cane roots in Java. Saccardo, in the Syll. fung., gives the habitat as 'in corticibus Sacchari officinarum'.

Clathrus columnatus Bosc. See Laternea columnata (Bosc) Nees.

Clathrus trilobatus Cobb. See Laternea columnata (Bosc) Nees.

Clavaria Wakkeri Sacc. & Syd.
*Wakker (C. gracillima), Ziekt. Suik., p. 195. 1898.
*Saccardo & Sydow (C. Wakkeri), Syll. fung., 14:237. 1899.
(Wakker's name pre-empted by Peck, 28th Report of the State Botanist of New York, p. 53. 1876).
On dead leaf sheaths in Java.

Coleroa Sacchari v. Breda. See Eriosphaeria Sacchari (v. Breda) Went.

Colletotrichum falcatum Went *Red Rot*
*Went, Archiev, 1:271. 1893.
A parasite in cane sticks, reported from Java, British West Indies, Queensland, India, Hawaii, Mauritius, Reunion, Louisiana, Brazil, Santo Domingo, Porto Rico, Cuba, the Philippines, and probably Argentina. Massee considers this fungus, together with Melanconium Sacchari and Thielaviopsis paradoxa, to be forms of Trichosphaeria Sacchari.

Coniosporium arundinis (Cda.) Sacc.
*Corda (Gymnosporium), Ic. fung., 2:1. 1838.
*Saccardo (Coniosporium), Michelia, 2:124. 1882.
Corda gives the habitat as 'auf faulenden Rohr- und Gras-holmen', while Saccardo mentions cane in particular.
Coniosporium extremorum Syd.
*Sydow, Ann. Myc., 11:270. 1913.
Saprophytic in the Philippines.
Coniosporium Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :248. 1896.
Occurs on the inner surface of sheaths at partly rotted internodes, in Argentina.
Coniosporium vinosum (B. & C.) Sacc.
Berkeley & Cooke (Gymnosporium), Fungi of Ceylon, No. 586.
*Saccardo (Coniosporium), Syll. fung., 4:244. 1886.
On dead leaves and culms in the Philippines and Ceylon. The above is the reference as given by Saccardo. There is no 'B. & C., Fungi of Ceylon' that can be located. The species does not appear in 'B. & Br., Fungi of Ceylon.'
Coniothyrium melasporum (Berk.) Sacc.
Berkeley (Darluca), Prae. Monogr. Hend., p. 26. 1878.
*Saccardo (Coniothyrium), Syll. fung., 3:319. 1884.
An imperfect form of Thyridaria tarda Bancr.
Coniothyrium Sacchari (Mass.) Prill. & Delacr. See Melanconium Sacchari Mass.
Corethropsis elegans Speg.
Spegazzini, Rev. Agr. Vet., :245. 1896.
Saprophytic, possibly weakly parasitic, on leaves still in the spindle, which have been weakened from some other cause, and on half rotted sheaths on the sticks, in Argentina.
Coriolopsis occidentalis (Klotzsch) Murr. See Polyporus occidentalis Klotzsch.
Corticium albido-carneum Rav. See Asterostroma albido-carnea (Schwein.) Mass.
Corticium arachnoideum Berk.
Berkeley, Outl., p. 273. 1860.
On cane trash and soil in Porto Rico.
Corticium cervicolor B. & C. See Asterostroma cervicolor (B. & C.) Mass.
Corticium cinereum Fr. See Peniophora cinerea (Fr.) Cke.
Corticium sp.
Undetermined species on dead cane in Porto Rico.
Craterium aureum (Schum.) Rost.
Schumacher (Trichia), En. pl. Saell, 2:421. 1803.
Rostafinski (Craterium), Sluzow. Monogr., p. 124. 1875.
On trash in Porto Rico and Santo Domingo.
Craterium leucocephalum (Pers.) Rost.
Persoon (Stemonites), in Syst. nat., 1:464. 1791.
Rostafinski (Craterium), Sluzow. Monogr., p. 123. 1875.
On trash in Porto Rico.
Crepidotus sp.
Undetermined species on rotted cane sticks in Porto Rico.
Cyathus Poeppigii Tul.
Tulasne, Ann. Sc. Nat. :76. 1844.
On trash in Porto Rico.
Cytospora Sacchari Butl.
*Butler, Mem. Dep. Agr. India, 1:(3):30. 1906.
Attacks the sticks and leaf sheaths of cane in India, Cuba, Porto Rico and Brazil.

D

Dacromyces Sacchari B. & Br.
*Berkeley & Broome, Trans. Linn. Soc. Lond., (2)2:65. 1883.
On charred cane sticks in Queensland.

Darluca melaspora Berk. See Coniothyrium melasporum (Berk.) Sacc.
Darluca melaspora, as identified by Prillieux and Delacroix is identical with Melanconium Sacchari.

Dasyscypha sp.
Undetermined species on cane trash in Porto Rico.

Dematium herbarum Pers. See Cladosporium herbarum (Pers.) Lk.

Dematium levisporum Speg.
Spegazzini, An. Mus. Nac. B. A., 20:433. 1910.
On rotting cane sticks in Argentina.

Dendrophoma saccharicola Averna.
*Averna-Sacca, Bol. Agric., 17:637. 1916.
Parasitic in the interior of the stick, in Brazil.

Depazea Sacchari Berk.
Reported saprophytic on cane leaves in Queensland. Original description cannot be located.

Diaporthe Sacchari Speg.
*Spegazzini, An. Mus. Nac. B. A., 19:364. 1909.
On rotted cane sticks in Argentina.

Dictydium cancellatum (Batsch) Macbr.
On trash in Porto Rico. No data can be located.

Dictyophora phalloidea Desv.
Desvaux, Jour. de Bot., 2:88. 1809.
A universally distributed saprophyte. At one time thought to be parasitic on cane roots in Hawaii.

Didymosphaeria saccharicola Speg.
*Spegazzini, An. Mus. Nac. B. A., 19:370. 1909.
On rotting cane sticks in Argentina.

Dinemasporium Sacchari Henn.
*Hennings, Hedwigia, 44:71. 1905.
Reported on cane leaves in Peru. Possibly a variety of D. gramineum Lév.

Dioranotropis vastatrix.
Parasitic in Reunion (according to the Revue Agricole, Reunion, No. 6, pp. 1-15, 1900, cited in the Experiment Station Record, Washington, U. S. A., 12:261, 1900-'01.) No further data concerning the fungus can be located.

Diplodia cacaoicola Henn.
*Hennings, Engl. Jahrb., 22:80. 1895.
An imperfect form of Thyridaria tarda Bancr.

E

Epicoccum levisporum Pat.
*Patouillard, Bull. Soc. Myc. France, 9:164. 1893.
On cane leaves in Ecuador.

Epicoccum purpurascens Ehrbg.
Ehrenberg, Sylv. myc. berol., p. 12. 1818.
Saprophytic on dead plants of various kinds, including Saccharum officinarum, in Europe.

Epicoccum vulgare Cda.
*Corda, Ic. fung., 1:5. 1837.
Habitat given by Corda as 'in caulibus herbarum putridis'. Saccardo mentions sugar cane specifically.

Eriosphaeria Sacchari (v. Breda) Went
van Breda de Haan (Coleroa), Mededeel. Suiker. p. 22. 1892.
*Went (Eriosphaeria), Ziekt. Suik., p. 155. 1898.
Parasitic on cane leaves in Java and Trinidad.

Erysiphe graminis DC.
De Candolle, Flore Fr., 5:106. 1815.
Occurs on various grasses, including species of Saccharum, in Europe, Asia, America and Australia.

Eurotium argentinum Speg.
Spegazzini, Rev. Agr. Vet., :228. 1896.
Described from drooping and partly decayed spindles of cane in Argentina. Reported from dead leaves and stalks, particularly museum specimens, in Porto Rico.

Euryachora Sacchari Averna
*Averna-Sacca, Bol. Agric., 17:618. 1916.
Parasitic in the iuterior of the stick, in Brazil.

Eutypella Sacchari Speg.
Spegazzini, An. Mus. Nac. B. A., (2)3:244. 1899.
Occurs on piles of rotting cane leaves in Argentina.

F

Fuligo septica (Linn.) Gmel.
Linnaeus (Mucor), Sp. plant., 2:No. 1656. 1753.
*Gmelin (Fuligo), Syst. nat., (13)2:1466. 1791.
Reported on cane trash in Porto Rico.

Fumago Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :256. 1896.
Reported on cane sticks in Argentina.

Fusarium spp.
Fusaria have been reported from various countries as saprophytic or doubtfully parasitic on sugar cane.

G

Gibbera Saubinetii Mont. See Gibberella Saubinetii (Mont.) Sacc.

Gibberella pulicaris (Fr.) Sacc.
*Fries (Sphaeria), Syst. myc., 2:417. 1822.
*Saccardo (Gibberella), Michelia, 1:317. 1879.
Reported on dead cane in Porto Rico.

Gibberella Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :237. 1896.
Occurs on the leaves and sometimes on the sticks of cane which has been killed by cold in Argentina.

Gibberella Saubinetii (Mont.) Sacc.
*Montagne (Gibbera), Syll. cryptog. No. 898, p. 252. 1856.
*Saccardo (Gibberella), Michelia, 1:513. 1879.
Montagne's description gives the fungus as saprophytic on numerous monocotyls in Europe and America. Saccardo mentions sugar cane specifically.

Glenospora Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :248. 1896.
Occurs on dead cane sticks in Argentina.

Gnomonia Iliau Lyon *Iliau*
*Lyon, H. S. P. A., No. 11, p. 28. 1912.
A sheath parasite, endemic to Hawaii and known for many years. Reported in 1913 from Louisiana as a probable immigrant.

Graphium nodulosum March.
Marchal, Bull. Soc. Belge Microsc., 20:266. 1894.
Reported on dead cane sticks in the lower Congo.

Graphium pistillarioides Speg.
Spegazzini, Rev. Agr. Vet., :254. 1896.
Occurs on rotting cane leaves in Argentina.

Graphium Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :253. 1896.
On rotting cane leaves and sheaths in Argentina, Porto Rico and Santo Domingo.

Graphium spp.
Undetermined species occur on cane leaves in Hawaii, about the wounds caused by leaf-hoppers (Perkinsiella saccharicida).

Guepinia palmiceps Berk.
*Berkeley, Ann. Mag. Nat. Hist., (1)10:383. 1842.
Occurs on dead cane sticks in Porto Rico.

Guepinia spathulata Jungh.
Junghuhn (Cantharellus), An. Sc. Nat., :320. 1841.
On dead cane sticks in Porto Rico.

Gymnoascus Reessii Baran.
*Baranetzky, Bot. Zeit., 30:158. 1872.
Occurs as a saprophyte on rotting cane sticks in Java.

Gymnosporium arundinis Cda. See Coniosporium arundinis (Cda.) Sacc.

Gymnosporium vinosum B. & C. See Coniosporium vinosum (B. & C.) Sacc.

H

Hansenia apiculata (Reess) Lind. var. **Sacchari** Racib.
Reess (Saccharomyces), Bot. Unter. Alk.-Pilze, p. 84. 1870.
Lindner (Hansenia),
*Raciborski, Archiev, 6:484. 1898.
A facultative parasite in Java. Lindner's description cannot be located.

Haplographium chlorocephalum (Fres.) Grove
Fresenius (Periconia), Beitr. Mykol., 1:21. 1850.
*Grove (Haplographium), Hardw. Sc. Goss., 21:198. 1885.
Occurs on rotting cane sticks in Madeira.

Haplographium Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :249. 1896.
On dead and dying leaves and sheaths in Argentina.

Haplosporella melanconioides Sacc.
Saccardo, Atti Ven.-Trent. Sc. Nat., 10:73. 1917.
On dead culms in the Philippines.

Helminthosporium Sacchari Butl. *Eye Spot*
*Butler, Mem. Dep. Agr. India, 6:207. 1913.
A leaf parasite in India, Cuba, Santo Domingo and Porto Rico. Possibly reported as Cercospora Sacchari from Hawaii, Java, Reunion, Jamaica and the Philippines.

Hendersonia Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :241. 1896.
Occurs on dying leaves and occasionally on sheaths in Argentina.

Hendersonina Sacchari Butl. *Collar Rot*
*Butler, Mem. Dep. Agr. India, 6:198. 1913.
Parasitic on cane sticks in India.

Himantia guttulifera Speg.
Spegazzini, Rev. Agr. Vet., :257. 1896.
On the inner side of dead sheaths in Argentina.

Himantia Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :257. 1896.
On rotting cane sticks which have been killed by cold in Argentina.

Himantia stellifera Johns. *Stellate-crystal Fungus*

*Johnston, Jour. Dep. Agr. P. R., 1:188. 1917.

Doubtfully parasitic, described from Porto Rico. What is apparently this same fungus has been reported from Hawaii, British Guiana, St. Croix and Jamaica, where it was supposed to be a form of Marasmius.

Hormiactella Sacchari Johns.

*Johnston, Jour. Dep. Agr. P. R., 1:224. 1917.

Occus on dead leaves in Santo Domingo and Porto Rico, often associated with withertip. Johnston believes that this fungus has been reported from Hawaii (H. S. P. A., No. 6:32. 1909.), but no name was suggested.

Hydnum Sacchari Spreng.

Sprengel, K. Svenska Vet. Akad. Handl., :21. 1820.

On dead cane leaves in Guadeloupe and Porto Rico.

Hypochnus Sacchari Speg.

Spegazzini, Rev. Agr. Vet., :227. 1896.

On drooping cane spindles in Argentina. Develops only on the growing part of cane diseased by Polvillo.

Hypocrea gelatinosa Tode. See Chromocrea gelatinosa (Tode) Seav.

Hypocrea rufa (Pers.) Fr.

Persoon (Sphaeria), Syn. meth. fung., p. 13. 1801.

Fries (Hypocrea), Summa veg. Scand., p. 383. 1846.

On dead cane sticks in Porto Rico.

Hypocrea saccharalis Racib.

*Raciborski, Par. Alg. Pil. Javas, 3:21 and 43. 1900.

On cane leaf sheaths in Java.

Hypocrea Sacchari Went

*Went, Archiev, 1:455. 1893.

On cane leaves and sheaths in Java.

Hypocrea saccharina Racib. See Hypocrea saccharalis Racib.

I

Ithyphallus celebicus Henn. *Stink-horn*

Hennings, Monsunia, 1:21. 1899.

Reported from Java as a possible parasite on cane roots. Probably merely saprophytic.

Ithyphallus coralloides Cobb. See Ithyphallus rubicundus (Bosc) Fisch.

Ithyphallus rubicundus (Bosc) Fisch. *Stink-horn*

Bosc (Satyrus), Mag. Ges. nat. Freunde, 5:86. 1811.

*Fischer (Ithyphallus), Syll. fung., 7:11. 1888.

A universal feeder on decaying vegetable matter. Described by Cobb (H. S. P. A., No. 5 :208. 1906) as a new species, I. coralloides, parasitic on cane roots in Hawaii. Since shown to be strictly saprophytic.

L

Lachnea cubensis (B. & C.) Sacc.

*Berkeley & Curtis (Peziza), Jour. Linn. Soc., 10:366. 1869.

*Saccardo (Lachnea), Syll. fung., 8:176. 1889.

On dead cane leaves in Porto Rico.

Lasiodiplodia Theobromae (Pat.) Griff. & Maubl.

*Patouillard (Botryodiplodia), Bull. Soc. Myc. Fr., 8:136. 1892.

*Griffon & Maublanc, Bull. Soc. Myc. France, 25:57. 1909.

An imperfect form of Thyridaria tarda Bancr.

Lasiosphaeria sp.

Undetermined species on dead cane in Porto Rico.

Laternea columnata (Bosc) Nees

Bosc (Clathrus), Mag. Ges. nat. Freunde, 5:85. 1811.

Nees von Esenbeck (Laternea), Syst. Pilze, p. 96. 1858.

Common in Florida and Louisiana, apparently attacking sick cane. One specimen found in Hawaii, growing on trash, and described by Cobb as Clathrus trilobatus, supposedly parasitic on cane roots. The fungus as a rule is a saprophyte.

Lentinus crinitus (Linn.) Fr.

Linnaeus (Agaricus), Sp. plant., 2:1644.

Fries (Lentinus), Acta R. Soc. Sci. Upsala, (3)1:34. 1851.

On dead cane sticks in Porto Rico.

Lepiota lycoperdinea Speg.

Spegazzini, An. Mus. Nac. B. A., (2)3:87. 1899.

On plowed land between cane rows in Argentina.

Leptosphaeria Sacchari v. Breda *Ring Spot*

van Breda de Haan, Mededeel. Suiker., 2:25. 1892.

(Description reprinted in Bijl. Archiev, :101. 1893).

Attacks cane leaves in Java, India, British West Indies, Hawaii, Brazil, Porto Rico, Argentina, Cuba and Santo Domingo.

Leptosphaeria Sacchari Speg. (non v. Breda). See Leptosphaeria Spegazzinii Sacc. & Syd.

Leptosphaeria saccharicola Henn.

*Hennings, Hedwigia, 39:79 (Beiblatt). 1900.

Parasitic on cane leaves in Brazil.

Leptosphaeria Spegazzinii Sacc. & Syd.

Spegazzini (L. Sacchari), Rev. Agr. Vet., :232. 1896.

*Saccardo & Sydow (L. Spegazzinii), Syll. fung., 14:570. 1899.

A facultative parasite on cane leaves in Argentina.

Leptosphaeria Spegazzinii Sacc. & Syd. var. **minor** Speg.

*Spegazzini, An. Mus. Nac. B. A., (3)12:383. 1909.

On cane sticks in Argentina.

Leptosphaeria tucumanensis Speg.

Spegazzini, Rev. Agr. Vet., :232. 1896.

Occurs on cane sticks weakened by disease but still alive, in Argentina.

Leptosphaeria sp. *Giant Ring Spot*

Parasitic on cane leaves in Hawaii. Apparently a new species, on this host plant at least, but not yet named.

Linospora Sacchari Averna

*Averna-Sacca, Bol. Agric., 17:614. 1916.

Parasitic on cane sticks in Brazil.

Lisea australis Speg. var. Sacchari Speg.

*Spegazzini, An. Soc. Cient. Arg., 12:210. 1881 (species).

Spegazzini, Rev. Agr. Vet., :236. 1896 (variety).

On rotting cane sticks in Argentina.

Lophodermium Sacchari Lyon

*Lyon, Hawaiian Pl. Rec., 9:601. 1913.

The fruiting bodies of this fungus look like short, raised, black lines running lengthwise of the midribs and leaf sheaths. When examined under a hand-lens they look like tiny black slugs (Fig. 1). As a rule they are nearly or quite straight, but sharply bent ones are occasionally met with, while branched ones are also sometimes found.

This fungus forms its fructifications within or beneath the epidermis, and as they develop the cuticle of the host is elevated into sharp ridges, which eventually split along their crests to expose the black fruit bodies. Upon exposure the fruit bodies in turn open a longitudinal slit along their crests to allow the escape of the spores which have been formed within. The spore-producing apparatus consists of club-shaped bags or asci (Fig. 3a), in each of which are produced eight slender, colorless spores (Fig. 3b). Associated with the asci are numerous slender, bulb-tipped filaments, the paraphyses, some of which are branched. This fungus is a new species belonging to the genus *Lophodermium* and may be technically described as follows:

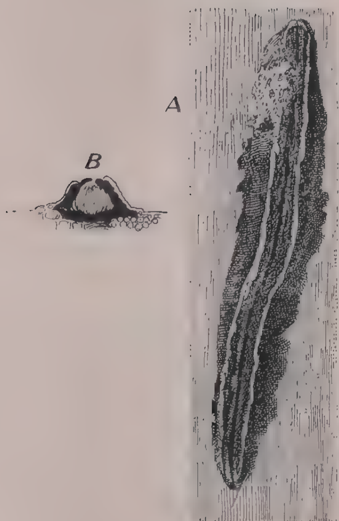


Fig. 1. *Lophodermium Sacchari.*
A, A single fructification viewed from above; B, cross section of the same; x33.

Fig. 2. *Lophodermium Sacchari.*
Cross section of fruit body showing asci, x180.

Fig. 3. *Lophodermium Sacchari.*
a, Asci and paraphyses; b, spores.

Perithecia black, at first sub-epidermal then erumpent, 1-3 rarely 4 mm. long, 1/4-1/2 mm. wide, longitudinally dehiscent.

Asci club-shaped, thin-walled, 85-100 x 4-5 μ.

Spores colorless, needle-shaped, 48-55 x 1-1.5 μ.

Habitat: On dead leaves of *Saccharum officinarum,* Hawaii.

Lophodermium sp.

Undetermined species on dead leaf sheaths in Porto Rico.

Lycogala epidendrum (Linn.) Fr.

*Linnaeus (Lycoperdon), Sp. plant., 2:1184. 1753.

Fries (Lycogala), Epicr., p. 80. 1836-1838.

On dead cane sticks in Porto Rico and Santo Domingo.

Lycoperdon albidum Cke.

Cooke, Jour. Roy. Micros. Soc., :723. 1887.

On cane trash in Porto Rico. Saccardo (Syll. fung.), spells the specific name 'albinum'.

Lycoperdon cinereum Batsch. See Physarum cinereum (Batsch) Pers.

Lycoperdon epidendrum Linn. See Lycogala epidendrum (Linn.) Fr.

Lycoperdon pusillum Batsch

Batsch, Elench. fung., 2: 1783-1789.

On trash in Porto Rico. Stevenson credits the species to Fries.

Lycoperdon pyriforme Schaeff.

Schaeffer, Icon. descr. fung., t. 189. 1761.

In Porto Rican cane fields.

M

Macrophoma Sacchari (Cke.) Berl. & Vogl.

Cooke, (Sphaeropsis), Grevillea, 12:23. 1884.

Berlese & Voglino, (Macrophoma), Atti Ven.-Trent. Sc. Nat., 10:186. 1886.

On cane sticks in Georgia, U. S. A.

Macrosporium graminum Cke.
Cooke, in Ravenel's American Fungi, No. 606, in Grevillea, 6:129 or 7:32,43. 1878.
Described from bamboo leaves in South Carolina. Reported from sugar cane in Tasmania.

Marasmius bambusinus Fr.
*Fries, Linnaea, 5:507. 1830. (as Agaricus).
On dead cane in Cuba.

Marasmius borinquensis Stevn.
*Stevenson, Jour. Dep. Agr. P. R., 1:218. 1917.
On cane in Porto Rico.

Marasmius Hiorami Murr.
*Murrill, N. Am. Fl., 9:256. 1915.
On dead sheaths and trash in Porto Rico.

Marasmius plicatus Wakk.
*Wakker, Ziekt. Suik., p. 195. 1898.
Occurs on dry and dead cane leaves in Java. Reported as parasitic from Louisiana and Porto Rico, where it is probably confused with M. stenophyllus. Reported from the Philippines.

Marasmius Sacchari Wakk. *Root Disease*
*Wakker, Centr. Bakt., (2)2:44. 1896.
Parasitic in Java, Hawaii, British West Indies, Mauritius, British Guiana, Santo Domingo, Cuba and Porto Rico.

Marasmius Sacchari Wakk. var. *hawaiiensis* Cobb, is identical with the species.

Marasmius semiustus Mass. See Marasmius stenophyllus Mont.

Marasmius stenophyllus Mont. *Banana Marasmius*
Montagne, Ann. Sc. Nat., (4)1:116. 1854.
Parasitic in Cuba, acting like M. Sacchari.

Marasmius synodicus (Kze.) Fr.
*Kunze (Agaricus), Linnaea, 5:507. 1830.
Fries (Marasmius), Epicr., p. 381. 1838.
Occurs on trash in Porto Rico.

Marasmius spp.
Several undetermined species on trash in Porto Rico.

Marsonia sp.
Undetermined species on midribs of dead cane leaves in Porto Rico. Indistinguishable macroscopically from Melanconium saccharinum.

Melanconium Iliau Lyon *Iliau*
*Lyon, Hawaiian Pl. Rec., 3:148. 1910.
An imperfect form of Gnomonia Iliau Lyon.

Melanconium lineolatum Sacc.
Saccardo, Atti Ven.-Trent. Sc. Nat., 10:83. 1917.
On dead cane leaves in the Philippines.

Melanconium Sacchari Mass. *Rind Disease*
*Massee, Ann. Bot., 7:515. 1893, and Kew Bull. No. 89. 1893. (No technical diagnosis in either).
Said by Massee to be parasitic on cane sticks in the West Indies. Saprophytic, or possibly very weakly parasitic, in nearly all sugar-growing countries. Thought by Massee to be a form of Trichosphaeria Sacchari Mass.

Melanconium saccharinum Penz. & Sacc.
*Penzig & Saccardo, Malpighia, 15:238. 1901.
Weakly parasitic on drooping cane leaves in Java, Porto Rico, Cuba and Louisiana. Generally a saprophyte, growing on dead leaves.

Melanospora globosa Berl.
*Berlese, Malpighia, 5:409. 1891.
On dead cane in Argentina.

Meliola Arundinis Pat.
*Patouillard, Journal de Bot., 11:348. 1897.
Occurs on the leaves of Saccharum spp. in the Philippines.

Merulius byssoides Burt
*Burt, Ann. Mo. Bot. Gard., 4:358. 1917.
On dead cane and trash, and on soil surrounding, in Porto Rico.

Metasphaeria sacchariсola Speg.
*Spegazzini, An. Mus. Nac. B. A., (3)10:376. 1909.
On rotting cane sticks in Argentina.

Metasphaeria Usteri Speg.
Spegazzini, Rev. Mus. La Plata, 15:23. 1908.
Occurs on drooping cane leaves in Brazil.

Microsphaeropsis Bakeri Syd.
Sydow, Ann. Myc., 14:369. 1916.
On culms of cane in the Philippines.

Microdiplodia melaspora (Berk.) Griff. & Maubl.
Berkeley, (Darluca), Prae. Monogr. Hend., p. 26.
*Griffon & Maublanc, Bull. Soc. Myc. Fr., 25:55. 1909.
An imperfect form of Thyridaria tarda Bancr.

Microtypha sacchariсola Speg.
*Spegazzini, An. Mus. Nac. B. A., (3)13:432. 1910.
On rotting cane sticks in Argentina.

Monilia sitophila (Mont.) Sacc.
Montagne (Penicillium), Centr. Bakt., (1)4:
*Saccardo (Monilia), Michelia, 2:359. 1882.
A saprophyte, growing over cane stubble in Hawaii and Porto Rico. Rare in fields which have not been burnt over. Occurs in Hawaii on press-cake which has been spread on the fields.

Mucor septicus Linn. See Fuligo septica (Linn.) Gmel.

Mycosphaerella striatiformans Cobb. See Sphaerella striatiformans (Cobb), Sacc. & Trott.

Myrothecium Verrucaria (Alb. & Schw.) Ditm.
*de Albertini & de Schweiniz (Peziza), Conspec. fung., p. 340. 1805.
*Ditmar (Myrothecium), Deutsch. Fl., 1:7. 1816.
On dead and dying cane sheaths in Porto Rico.

N

Nectria flavociliata Seav.
*Seaver, Mycologia, 1:54. 1909.
On dead cane sticks in Porto Rico.

Nectria Laurentiana March.
Marchal, Bull. Soc. Belge Microsc., 20:261. 1894.
On dying cane sticks in Santo Domingo, Porto Rico and the Congo.

Nectria sacchariсola Speg.
Spegazzini, Rev. Agr. Vet., :234. 1896.
On rotting cane sticks in Argentina.

Nectria spp.
Undetermined species reported saprophytic from Hawaii, Porto Rico and Brazil.

O

Odontia Sacchari Burt
*Burt, Ann. Mo. Bot. Gard., 4:233. 1917.
On dead sheaths and sticks in Porto Rico.

Odontia sacchariсola Burt — *Granular Leaf-Sheath Fungus*
*Burt, Ann. Mo. Bot. Gard., 4:235. 1917.
On dead leaf sheaths in Santo Domingo and Porto Rico. May be the perfect stage of Himantia stellifera.

Odontia sp.
Undetermined species on dead cane sticks in Porto Rico.

Oedocephalum Bergii Speg.
Spegazzini, Rev. Agr. Vet., :244. 1896.
A facultative parasite, occurring sometimes on living cane, but generally found on rotting sticks, in Argentina.
Oospora tomentella Speg.
Spegazzini, Rev. Agr. Vet., :243. 1896.
In diseased spindles of young plant cane in Argentina.
Ophiognomonia Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :231. 1896.
On drooping leaves and sheaths in Argentina.
Otthia sp.
One undetermined species reported parasitic in Brazil.
Ozonium Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :257. 1896.
On dry sheaths in Argentina.

P

Penicillium glaucum Link
Link, Mag. Ges. nat. Freunde, 3:15. 1809.
Reported saprophytic in Java.
Penicillium platense Speg.
Spegazzini, Rev. Agr. Vet., :246. 1896.
On the inner surface of sheaths at partly rotted internodes, in Argentina.
Penicillium sitophilum Mont. See Monilia sitophila (Mont.) Sacc.
Peniophora cinerea (Fr.) Cke.
Fries (Corticium), Epicr., p. 563. 1838.
Cooke (Peniophora), Grevillea, 8:20. 1879.
On dead cane sticks in Porto Rico.
Peniophora flavido-alba Cke.
Cooke, Grevillea, 8:21. 1879.
On dead cane sticks in Porto Rico.
Peniophora spp.
Undetermined species reported saprophytic from Cuba and Porto Rico.
Periconia Sacchari Johns.
*Johnston, Jour. Dep. Agr. P. R., 1:225. 1917.
On dead and dying cane leaves in Porto Rico, often associated with wither-tip. A weak parasite.
Pestalozzia fuscescens Sor. var. **Sacchari** Wakk.
Sorauer, Handb. Pflanzenkr., 2:400. 1886. (species).
*Wakker, Ziekt. Suik., p. 163. 1898. (variety).
A leaf parasite in Java.
Peziza cubensis B. & C. See Lachnea cubensis (B. & C.) Sacc.
Peziza Verrucaria Alb. & Schw. See Myrothecium Verrucaria (Alb. & Schw.) Ditm.
Phaeoporus luteoumbrinus Rom.
Romell, K. Svenska Vet. Akad. Handl., 24:27. 1901.
Occurs on old cane roots in Argentina. A weak parasite.
Phallus aurantiacus (Mont.) Fisch. See Ithyphallus rubicundus (Bosc) Fisch.
Phoma heterospora Speg.
Spegazzini, Rev. Agr. Vet., :239. 1896.
In cane spindles killed by cold, in Argentina.
Phyllachora Sacchari Henn.
*Hennings, Hedwigia, 41:143. 1902.
Parasitic on cane leaves in Java and the Philippines.

Phyllosticta hawaiiensis Caum

*Caum, Hawaiian Pl. Rec., 20:278. 1919.

Fig. 1. Spores of *P. hawaiiensis.*

Fig. 2. Pycnidium seen from above.

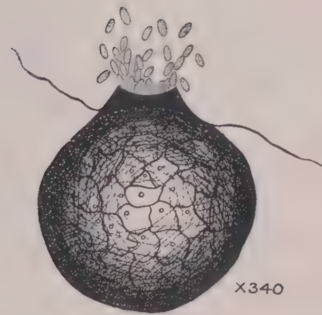


Fig. 3. Pycnidium discharging spores.

This fungus, which seems to attack practically all of the cane varieties grown in Hawaii, is primarily a leaf-sheath parasite, attacking the sheath at or near the point of attachment, killing the tissues and causing the formation of characteristic spots. In many cases, however, the fungus does not limit its growth to the sheath, but pushes downward onto the rind of the internode below. In one or two particularly susceptible varieties cases have been found in which all the rind on the internode has been destroyed. The ordinary appearance of the fungus, however, is a small spot on the sheath, which can easily escape observation. This fungus, while rather widely spread in Hawaii, is not common in any one place, and is probably of very minor importance. A technical diagnosis follows:

Spots conspicuous, dry, straw-colored, sharply defined, sometimes margined with dark red to purple; pycnidia brown, reticulated, at first submerged, later erumpent, scattered in the spots, roughly globose, with one, occasionally two and rarely three slightly papillate ostioles, which are suituated in the center of a narrow black area, size ranging from 30 x 30 to 168 x 136 μ in single-ostioled specimens to 191 x 141 in double-ostioled specimens; spores elliptical, minute, 3.2 to 6.5 x 1.3 to 2.8 μ, hyaline, smooth, with two and sometimes three vacuoles. The pycnidia developed in the rind average much smaller than those developed in the sheaths. Parasitic in the leaf-sheath and rind of the sugar cane, *Saccharum officinarum,* in Hawaii.

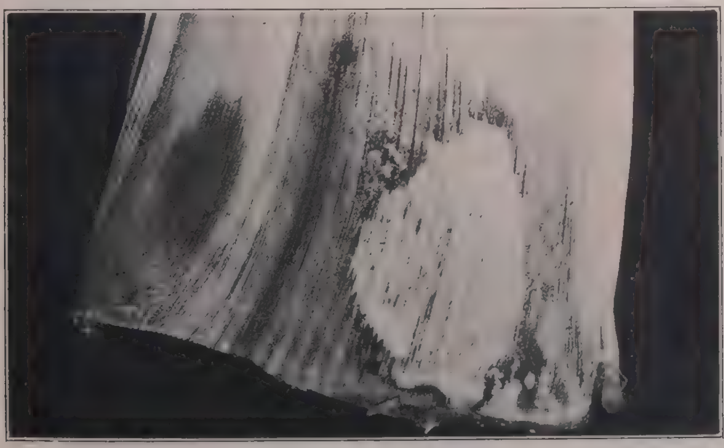


Fig. 4. *Phyllosticta hawaiiensis.* A characteristic spot on the leaf-sheath, enlarged.

Phyllosticta Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :239. 1896.
On cane leaves in Argentina, Brazil (and Porto Rico?)

Phyllosticta saccharicola Henn.
*Hennings, in Fl. B. et M. Congo, 2:100. 1907.
On cane leaves in the Congo.

Phyllosticta sp.
An undetermined species reported parasitic on cane leaves in Brazil.

Physalospora paraguaya Speg.
*Spegazzini, An. Soc. Cient. Arg., 18:266. 1884.
On rotting sticks in Brazil.

Physalospora Sacchari (B. & Br.) Sacc.
*Berkeley & Broome (Sphaeria), Trans. Linn. Soc. Lond., (2)2:223. 1887.
*Saccardo (Physalospora), Syll. fung., 9:599. 1891.
On cane leaves in Queensland.

Physalospora tucumanensis Speg.
Spegazzini, Rev. Agr. Vet., :228. 1896.
On dead cane leaves, sheaths and sticks in Argentina and Porto Rico.

Physarum cinereum (Batsch) Pers.
Batsch (Lycoperdon), Elench. fung., 1783-1789.
Persoon (Physarum), Syn. meth. fung., p. 170. 1801.
On living leaves of various plants, including the sugar cane, in Porto Rico.

Physarum compressum Alb. & Schw.
*de Albertini & de Schweiniz, Conspec. fung., p. 97. 1805.
Found on cane leaves in Porto Rico.

Physarum didermoides (Ach.) Rost.
*Acharius (Spumaria), Syn. meth. fung. addenda, p. 29. 1801.
Rostafinski (Physarum), Sluzow. Monogr., p. 97. 1875.
Occurs on dead vegetation generally. Saccardo mentions sugar cane as a host.

Physarum globuliferum (Bull.) Rost.
Bulliard (Sphaerocarpus), Hist. Champ. Fr., p. 134. 1791-1798.
Rostafinski (Physarum), Sluzow. Monogr., p. 98. 1875.
Described from rotting vegetation in Europe. Saccardo mentions sugar cane as a host.

Physarum nodulosum (Cke. & Balf.) Mass.
Occurs on green sheaths in Porto Rico. No data can be located.

Physarum tucumanensis Speg.
Spegazzini, Rev. Agr. Vet., :237. 1896.
Occurs on rotting cane in Argentina.

Plasmodiophora vascularum Matz
*Matz, Jour. Dep. Agr. P. R., 4:45-6. 1920.
A vessel-plugging organism found in cane affected with Yellow Stripe disease, in Porto Rico.

Pleospora infectoria Fckl. var. **Sacchari** Speg.
Fuckel, Symb. mycol., p. 132. 1871-1872.
Spegazzini, Rev. Agr. Vet., :233. 1896.
Occurs on drooping leaves, rarely on old sheaths, in Argentina.

Polydesmus sp.
Undetermined species on dead leaves in Porto Rico. Not distinguishable macroscopically from Spegazzinia and Tetracoccosporis.

Polyporus nivosa Berk. See Trametes nivosa (Berk.) Murr.

Polyporus occidentalis Klotz.
Klotzsch, Linnaea, 8:486. 1833.
On dead cane sticks in Porto Rico.

Polyscytalum Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :243. 1896.
On young leaves which are drooping and sickly, in Argentina.

Polystictus sanguineus (Linn.) Mey.
Linnaeus (Boletus), Sp. plant., 2:1646.
Meyer (Polystictus), Prim. Fl. esseq., p. 304. 1818.
Found on dead cane sticks in Porto Rico. Reported on old but still living cane roots in Argentina by Spegazzini, who credits the species to Saccardo. May become at times a weak parasite.

Polystictus sinuosus (Fr.) Sacc.
On dead cane sticks in Porto Rico. Descriptions cannot be located.

Pseudomonas vascularum (Cobb) E. F. Sm. *Gummosis*
*Cobb (Bacillus), Rep. Dep. Agr. N. S. W., p. 8. 1893.
*E. F. Smith (Pseudomonas), Centr. Bakt., (2)13:731. 1904.
A cane parasite in New South Wales, Queensland, Fiji, Mauritius, Java, Borneo, New Guinea, Brazil, the West Indies and Reunion.

Pycnoporus sanguineus (Linn.) Murr. See Polystictus sanguineus (Linn.) Mey.

Pyrenium lignorum vulgare Tode. See Trichoderma lignorum (Tode) Harz

Pythium spp.
Several undetermined species reported as parasitic on the roots in Java and Hawaii.

R

Rheosporangium sp.
On cane roots in Hawaii.

Rhinocladium Sacchari Speg.
Spegazzini, Rev. Agr. Vet./ :250. 1896.
On the inner side of sheaths in Argentina.

Rhizoctonia sp.
On cane roots in Hawaii.

Rhizoctonia sp. *Blight*
On young leaves in the Philippines.

Rosellinia paraguayensis Speg.
*Spegazzini (Anthostomella), An. Soc. Cient. Arg., 18:267. 1884.
*Spegazzini (Rosellinia), An. Mus. Nac. B. A., (3)12:338. 1909.
On dead cane in Brazil, Argentina and Porto Rico. (Stevenson credits the species to Stark).

Rosellinia pulveracea (Ehrh.) Fckl.
Ehrhart (Sphaeria), Syn. meth. fung., p. 83. 1801.
Fuckel (Rosellinia), Symb. mycol., p. 149. 1869-1872.
On dead cane in Porto Rico.

Rostafinsckia australis Speg.
*Spegazzini, An. Soc. Cient. Arg., 10:151. 1880.
On rotting cane sticks in Argentina.

S

Satyrus rubicundus Bosc. See Ithyphallus rubicundus (Bosc) Fisch.

Schizophyllum alneum (Linn.) Schoet. See Schizophyllum commune Fr.

Schizophyllum commune Fr.
*Fries, Syst. myc., 1:333. 1821.
Generally saprophytic, but occasionally a parasite. Considered the cause of dry-rot in Porto Rico. Reported on cane in Java, West Indies, Brazil, Hawaii and the Philippines.

Schizophyllum lobatum Bref.
*Brefeld, Unters. Ges. Mykol., 8:67. 1889.
On dead cane in Java. (Saccardo, in Syll. fung., erroneously credits the species to Went.)

Scirrhia lophodermoides El. & Ev.
*Ellis & Everhart, Bull. Torr. Bot. Club, 22:435. 1895.
On dead cane in Porto Rico.

Sclerospora Sacchari Miy. *Downy Mildew, Dew Fungus*
*Miyake, Formosa Bull., No. 1:12-13. 1911.
Occurs as a parasite on cane leaves in Formosa and the Philippines.

Sclerospora sp. *Leaf-Splitting Fungus*

On cane leaves in the Philippines.

Sclerotium griseum Stevn. *Sclerotial Disease*

Stevenson, in ed. Mentioned Jour. Dep. Agr. P. R., 2:220. 1918.

Parasitic in Porto Rico and Santo Domingo.

Sclerotium Rolfsii Sacc. *Sclerotial Disease*

*Saccardo, Ann. Myc., 9:257. 1911.

A sheath parasite in Hawaii, Louisiana, Porto Rico, St. Croix, Cuba and the Barbados.

Sclerotium spp.

Several undetermined species parasitic in Hawaii, Fiji, Java and the Philippines. In Java they are known as 'zuur rot' and 'djamoer oepas'.

Scytinotus distantifolius Murr.

*Murrill, N. Am. Fl., 9:239. 1915.

On dead cane leaves in Porto Rico.

Septonema Sacchari J. & S.

*Johnston & Stevenson, Jour. Dep. Agr. P. R., 1:225. 1917.

On cane leaves in Porto Rico.

Spegazzinia ornata Sacc.

*Saccardo, Michelia, 2:172. 1882.

On dead cane leaves in Porto Rico.

Spegazzinia tucumanensis Speg.

Spegazzini, Rev. Agr. Vet., :256. 1896.

On rotting leaves and sheaths in Argentina.

Sphaerella Sacchari Speg.

Spegazzini, Rev. Agr. Vet., :30. 1896.

Occurs on cane spindles which have been killed by cold in Argentina. On dead leaves in Porto Rico, and in Cuba it is often associated with wither-tip.

Sphaerella Sacchari Wakk. (non Speg.) See Sphaerella Wakkeri Sacc. & Syd.

Sphaerella striatiformans (Cobb) Sacc. & Trott.

*Cobb (Mycosphaerella), H. S. P. A., No. 5:104. 1906.

*Saccardo and Trotter (Sphaerella), Syll. fung., 22:145. 1913.

On dead cane leaves in Hawaii. Said by Cobb to be the cause of the 'leaf-splitting disease', but since shown to be saprophytic.

Sphaerella Wakkeri Sacc. & Syd.

*Wakker, (S. Sacchari), Ziekt. Suik., p. 196. 1898.

*Saccardo & Sydow (S. Wakkeri), Syll. fung., 14:533. 1899.

On dead cane leaves in Java.

Sphaeria pulicaris Fries. See Gibberella pulicaris (Fr.) Sacc.

Sphaeria rufa Persoon. See Hypocrea rufa (Pers.) Fr.

Sphaerobolus stellatus Tode

*Tode, Fung. meck. sel., 1:43. 1790.

On rotting cane trash in Porto Rico.

Sphaerocarpus globulifer Bull. See Physarum globuliferum (Bull.) Rost.

Sphaeronema adiposum Butl. *Black Rot*

*Butler, Mem. Dep. Agr. India, 1:(3) :40. 1906.

A parasite in cane sticks in India. Possibly occurs in Louisiana also.

Sphaeropsis pseudo-diplodia (Fckl.) Delacr.

Fuckel (Diplodia), Symb. mycol., p. 393. 1869-1870.

*Delacroix (Sphaeropsis), Bull. Soc. Myc. Fr., 19:350. 1903.

Parasitic at the tip of the culm, in Brazil. Delacroix considers the fungus identical with Sph. malorum Peck.

Sphaeropsis Sacchari Cke. See Macrophoma Sacchari (Cke.) Berl. & Vogl.

Sphaerulina Sacchari Henn.

*Hennings, Hedwigia, 44:62. 1905.

On cane leaves in Peru.

Sporocybe Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :253. 1896.
On partly rotted cane in Argentina.

Sporodesmium Sacchari Speg.
*Spegazzini, An. Mus. Nac. B. A., (3)13:443. 1910.
On rotting cane in Argentina.

Sporoschisma paradoxum deSeynes. See Thielaviopsis paradoxa (deSeyn.) v. Höhn.

Spumaria didermoides Ach. See Physarum didermoides (Ach.) Rost.

Stachybotrys pulcra Speg.
Spegazzini, Rev. Agr. Vet., :248. 1896.
Occurs on stems and occasionally on drooping leaves, and on leaves which have been cut and bundled, but which are still living, in Argentina.

Stemonites fusca Roth
Roth, Fl. germ., 1:448. 1788.
On cane trash in Porto Rico.

Stemonites leucocephala Pers. See Craterium leucocephalum (Pers.) Rost.

Stemonites splendens Rost.
Rostafinski, Sluzow. Monogr., p. 195. 1875.
On cane trash in Porto Rico.

Sterigmatocystis nigra v. Tiegh.
van Tieghem, Bull. Soc. Bot. Fr., 27:30. 1880.
Saprophytic in Porto Rico, especially on imperfectly sterilized material in moist chambers.

Stigmella Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :251. 1896.
Occurs on cane sticks, sheaths and leaves, in Argentina.

Stigmina Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :251. 1896.
On drooping but still living cane leaves in Argentina.

Stigmina pulchella Speg.
Spegazzini, Rev. Agr. Vet., :251. 1896.
On sticks, sheaths and leaves in Argentina.

Stilbum incarnatum Wakk.
*Wakker, Ziekt. Suik., p. 197. 1898.
On rotting cane sticks in Java.

Stilbum proliferum March.
Marchal, Bull. Soc. Belge Microsc., 20:267. 1894.
On rotting cane sticks in the Congo.

Stilbum sp.
Undetermined species on dead cane sticks in Porto Rico.

Strumella Sacchari Cke. See Melanconium Sacchari Mass.

T

Tapesia sp.
Undetermined species on dead cane sticks in Porto Rico.

Tetracoccosporis Sacchari Stevn.
*Stevenson, Jour. Dep. Agr. P. R., 1:225. 1917.
On dead cane leaves in Porto Rico.

Tetraploa aristata B. & Br.
*Berkeley & Broome, Ann. Mag. Nat. Hist., (2)5:460. 1850.
On dead cane sticks in Porto Rico and Santo Domingo.

Thielaviopsis ethaceticus Went. See Thielaviopsis paradoxa (deSeyn.) v.Höhn.

Thielaviopsis paradoxa (de Seyn.), v. Höhn. *Pineapple Disease*
*de Seynes (Sporoschisma), Rech. Veg. Inf., 3:30. 1886.
*von Höhnel (Thielaviopsis), Hedwigia, 43:295. 1904.
A wound parasite, particularly on cuttings, in Java, India, Hawaii, British West Indies, British Guiana, Mauritius, Louisiana, Brazil, Santo Domingo, Porto Rico and the Philippines. Said by Massee to be a form of Trichosphaeria Sacchari.

Thyridaria tarda Bancr.
*Bancroft, Malay Bull., No. 9:26. 1911.
Parasitic on various plants, including sugar cane, in the Malay States, British West Indies, India, British Guiana, Ceylon, Brazil, Philippines, Java, Samoa, Porto Rico and tropical America and tropical Africa in general.

Trametes nivosa (Berk.) Murr.
Berkeley (Polyporus), Hook. Jour. Bot., 8:196. 1856.
*Murrill (Trametes), N. Am. Fl., 9:42. 1907.
On dead cane sticks in Porto Rico and Santo Domingo.

Trametes pusilla Racib.
*Raciborski, Archiev, 6:489. 1898.
A rare saprophyte in Java.

Treleasia Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :235. 1896.
On young spindles weakened by some other cause, in Argentina.

Treleasiella Sacchari Speg.
Spegazzini, Rev. Agr. Vet., :271. 1896.
On cane spindles in Argentina.

Tremellodendron simplex Burt
*Burt, Ann. Mo. Bot. Gard., 2:742. 1915.
On trash in Porto Rico.

Trichia aurea Schum. See Craterium aureum (Schum.) Rost.

Trichoderma lignorum (Tode) Harz
*Tode (Pyrenium), Fung. meck. sel., 1:33. 1790.
Harz (Trichoderma), Bull. Soc. Imp. Mosc., 44:29. 1871.
On dead cane in Porto Rico and Hawaii.

Trichosphaeria Sacchari Mass.
*Massee, Ann. Bot., 7:515. 1893.
A stem parasite in the British West Indies and Reunion. Massee claims this fungus to be the perfect stage of Melanconium Sacchari, Colletotrichum falcatum and Thielaviopsis paradoxa. These are, however, generally considered distinct.

Triposporium muricatum Wakk.
*Wakker, Ziekt. Suik., p. 197. 1898.
Occurs on dead cane leaves in Java.

Trogia sp.
An undetermined species on dead cane in Porto Rico.

Trullula Sacchari. See Melanconium Sacchari Mass.

Tubercularia saccharicola Speg.
Spegazzini, Rev. Agr. Vet., :254. 1896.
On dead sticks in Argentina, Santo Domingo and Porto Rico.

Thelephora albido-carnea Schwein. See Asterostroma albido-carneum (Schwein.) Mass.

Trichia cinerea Bull. See Arcyria cinerea (Bull.) Schum.

U

Uredo Kühnii (Krüg.) W. & W. See Uromyces Kühnii Krüger.

Uromyces Kühnii Krüger *Rust*
*Krüger, Ber. Zuck., 1:117. 1890.
A leaf parasite in Java, the Philippines, Japan, Australia and India. According to Butler, the genus should be Puccinia rather than Uromyces.

Ustilago Sacchari Rbh. *Smut*
Rabenhorst, Isis, 4: 1870.
A parasite on the sticks and spindles of various species of Saccharum, including S. officinarum, in Italy, Natal, India, Trinidad, Java, Reunion, China, Philippines, Mauritius, Queensland, and British Guiana.

V

Valsa sp.
An undetermined species reported saprophytic in Porto Rico.

Valsaria subtropica Speg.
 *Spegazzini, An. Mus. Nac. B. A., (3)10:372. 1909.
  On rotted cane sticks in Argentina, Porto Rico and Brazil.

Venturia Sacchari v. Breda
 *van Breda de Haan (Coleroa), Bijl. Archiev, p. 99. 1893.
  On cane leaves in Java.

Venturia sterilis Speg.
 Spegazzini, Rev. Agr. Vet., :230. 1896.
  On young cane spindles which have been weakened from some other cause, in Argentina.

Vermicularia graminicola West.
 Westendorp, Bull. Ac. Roy. Belge.
  On dead cane sticks in Porto Rico. Reference above as given in Syll. fung. There is no 'Bull. Ac. Roy. Belge' that can be located.

Vermicularia Sacchari Averna
 *Averna-Sacca, Bol. Agric., 17:629. 1916.
  Parasitic in the interior of the stick, in Brazil.

Verticicladium graminicolum J. & S.
 *Johnston and Stevenson, Jour. Dep. Agr. P. R., 1:226. 1917.
  On cane leaves in Porto Rico.

Verticillium glaucum Bon.
 *Bonorden, Handb. allg. Mykol., p. 97. 1851.
  Described from decayed vegetation in Europe. Saccardo mentions sugar cane as a host.

Verticillium Sacchari Went. See Hypocrea Sacchari Went.

Vibrio subtilis Ehrbg. See Bacillus subtilis (Ehrbg.) Cohn.

Volutina sp.
 An undetermined species on dead cane in Porto Rico.

X

Xiphomyces Sacchari Syd.
 *Sydow, Ann. Myc., 14:374. 1916.
  On leaf-sheaths in the Philippines.

Xylaria apiculata Cke.
 Cooke, Grevillea, 8:66. 1879.
  On dead cane sticks in Porto Rico.

Z

Zygodesmus guarapiense Speg.
 *Spegazzini, An. Soc. Cient. Arg., 22:209. 1886.
  On rotting cane sticks in Argentina.

The sugar-cane diseases listed below are still obscure, the causative organisms or conditions having not as yet been fully determined.

- *Yellow Stripe Disease, Mosaic Disease, Mottling Disease,* or *Gele Strepenziekte,* of practically all sugar-growing countries.
- *Infectious Top-Rot,* of Hawaii.
- *Pokkah Bong,* of Java.
- *Polvillo,* of South America.
- *Fiji Disease,* of Fiji, Australia, Papua and the Philippines.
- *Sereh,* of Java.
- *Lahaina Disease, Root Rot,* or *Rood Rot,* of Hawaii, the West Indies and Java.
- *Pahala Blight,* of Hawaii.

A General Classification of Sugar-Cane Fungi.

ORDER	FAMILY	GENUS
Schizomycetes	Bacteriaceae	Bacillus
		Bacterium
		Pseudomonas
	Myxobacteriaceae	Chondromyces
Myxomycetes	Arcyriaceae	Arcyria
	Brefeldiaceae	Rostafinsckia
	Myxomycetaceae	Craterium
		Dictydium
		Lycogala
		Stemonites
	Physaraceae	Fuligo
		Physarum
Phycomycetes	Peronosporaceae	Pythium
		Sclerospora
Ascomycetes	Capnodiaceae	Capnodium
	Dothideaceae	Euryachora
		Phyllachora
		Scirrhia
	Gymnoascaceae	Gymnoascus
	Heliotiaceae	Dasyscypha
	Hypocreaceae	Calonectria
		Chromocrea
		Gibberella
		Hypocrea
		Lisea
		Melanospora
		Nectria
		Treleasia
	Hysteriaceae	Lophodermium
	Mollisiaceae	Tapesia
	Perisporaceae	Asterula
		Erysiphe
		Eurotium
		Meliola
	Pezizaceae	Lachnea
	Saccharomycetaceae	Hansenia

ORDER	FAMILY	GENUS
Ascomycetes	Sphaeriaceae	Apiospora
		Chaetomium
		Diaporthe
		Didymosphaeria
		Eriosphaeria
		Eutypella
		Gnomonia
		Lasiosphaeria
		Leptosphaeria
		Linospora
		Metasphaeria
		Microsphaeropsis
		Ophiognomonia
		Otthia
		Physalospora
		Pleospora
		Rosellinia
		Sphaerella
		Sphaerulina
		Thyridaria
		Trichosphaeria
		Valsa
		Valsaria
		Venturia
		Xylaria
Basidiomycetes	Agaricaceae	Crepidotus
		Lentinus
		Lepiota
		Marasmius
		Schizophyllum
		Scytinotus
		Trogia
	Clavariaceae	Clavaria
	Hydnaceae	Hydnum
		Odontia
	Lycoperdaceae	Lycoperdon
	Nidulariaceae	Cyathus
	Phallaceae	Dictyophora
		Ithyphallus
		Laternea
	Polyporaceae	Merulius
		Phaeoporus
		Polyporus
		Polystictus
		Trametes

ORDER	FAMILY	GENUS
Basidiomycetes	Thelephoraceae	Asterostroma
		Cladoderris
		Corticium
		Hypochnus
		Peniophora
	Tremellaceae	Dacromyces
		Guepinia
		Tremellodendron
	Uredinaceae	Uromyces
	Ustilaginaceae	Ustilago
Fungi Imperfecti	Dematiaceae	Acremoniella
		Acrothecium
		Alternaria
		Arthrinium
		Basisporium
		Catenularia
		Cercospora
		Cladosporium
		Coniosporium
		Dematium
		Fumago
		Glenospora
		Haplographium
		Helminthosporium
		Hormiactella
		Macrosporium
		Microtypha
		Periconia
		Polydesmus
		Rhinocladium
		Septonema
		Sporodesmium
		Stachybotrys
		Stigmella
		Stigmina
		Tetracoccosporis
		Tetraploa
		Thielaviopsis
		Verticicladium
		Zygodesmus
	Excipulaceae	Dinemasporium
	Melanconiaceae	Colletotrichum
		Marsonia
		Melanconium
		Pestalozzia

ORDER	FAMILY	GENUS
Fungi Imperfecti	Moniliaceae	Acrostalagm
		Allantospor:
		Arthrobotry
		Aspergillus
		Botrytis
		Cephalosporium
		Corethropsis
		Monilia
		Oedocephalum
		Oospora
		Penicillium
		Polyscytalum
		Sterigmatocystis
		Trichoderma
		Verticillium
	Nidulariaceae	Sphaerobolus
	Phomataceae	Apiosphaeria
		Bakerophoma
		Chaetomella
		Chaetophoma
		Coniothyrium
		Cytospora
		Dendrophoma
		Depazea
		Diplodia
		Haplosporella
		Hendersonia
		Hendersonina
		Lasiodiplodia
		Macrophoma
		Microdiplodia
		Phoma
		Phyllosticta
		Sphaeronema
		Sphaeropsis
		Vermicularia
	Sterile Mycelia	Himantia
		Ozonium
		Sclerotium
	Stilbaceae	Graphium
		Sporocybe
		Stilbum

ORDER	FAMILY	GENUS
Fungi Imperfecti	Tuberculariaceae	Chaetostroma
		Epicoccum
		Fusarium
		Myrothecium
		Spegazzinia
		Tubercularia
		Volutina
		Xiphomyces
	Zythiaceae	Treleasiella
	Classification Unknown	Chromocreopsis
		Dioranotropis

The classification above is mainly after the 'Sylloge fungorum,' by P. A. Saccardo, and 'Genera of Fungi', by F. E. Clements.

Abbreviations Used in Citing Names of Publications.

A

Acta R. Soc. Sci. Upsala = Nova Acta Regiae Societatis Scientiarum Upsaliensis. Upsala (Sweden), 1773 - →

An. Mus. Nac. B. A. = Anales del Museo Nacional de Historia Natural. Buenos Aires (Argentina), 1864 - →

An. Soc. Cient. Arg. = Anales de la Sociedad Cientifico de Argentina. Buenos Aires (Argentina), 1876 - →

Ann. Bot. = Annals of Botany. London (England), 1887 - →

Ann. Mag. Nat. Hist. = Annals and Magazine of Natural History. London (England), 1829 - →

Ann. Mo. Bot. Gard. = Annals of the Missouri Botanical Garden. St. Louis (U. S. A.), 1914 - →

Ann. Myc. = Annales Mycologici. Berlin (Germany), 1903 - →

Ann. Sc. Nat. = Annales des Sciences Naturelles, Botanique. Paris (France), 1824 - →

Archiev = Archiev voor de Java-Suikerindustrie. Soerabaja (Java), 1893 - →

Atl. Prin. Bact. = Atlas and Principles of Bacteriology. (Translated from the German). Philadelphia (U. S. A.), 1901.

Atti Ven.-Trent. Sc. Nat. = Atti della Societa Veneto-Trentina di Scienze Naturale. Padua (Italy), 1874 - → (Now called Atti della Accademia Scientifica Veneto-Trentino-Istriana).

B

Beitr. Biol. = Cohn, Beiträge zur Biologie der Pflanzen. Breslau (Germany), 1875 - →

Beitr. Mykol. = Beiträge zur Mykologie. Frankfurt-am-Main (Germany), 1850 - 1852.

Ber. Zuck. = Berichte der Versuchsstation für Zuckerrohr in West-Java. Kagok-Tegal (Java), 1890 - 1892.

Bijl. Archiev = Bijlage van het Archiev voor de Java-Suikerindustrie. Soerabaja (Java), 1893 - →

Bol. Agric. = Boletim de Agricultura. Saõ Paulo (Brazil), 1900 - →

Bot. Unter. Alk.-Pilze = Botanische Untersuchungen über die Alkoholgährungspilze. Leipzig (Germany), 1870.

Bot. Zeit. = Botanisches Zeitung. Leipzig (Germany), 1843 - 1910.

Bull. Soc. Belge Microsc. = Bulletin de la Société Belge de Microscopie. Brussels (Belgium), 1875 - →

Bull. Soc. Bot. Fr. = Bulletin de la Société Botanique de France. Paris (France), 1854 - →

Bull. Soc. Imp. Mosc. = Bulletin de la Société Impériale des Naturalistes de Moscou. Moscow (Russia), 1829 - ?

Bull. Soc. Myc. France = Bulletin de la Société Mycologique de France. Lons-le-Saunier, Autun, Poligny and Paris (France), 1885 - →

Bull. Torr. Bot. Club = Bulletin of the Torrey Botanical Club. New York (U. S. A.), 1870 - →

C

Centr. Bakt. = Centralblatt für Bakteriologie und Parasitenkunde. 2nd series. Jena (Germany), 1895 - →

Conspec. fung. = Conspectus fungorum in Lusatiae superioris agro Niskiensi crescentium. Lipsiae (= Leipzig, Germany), 1805.

D

Deutsch. Fl. = Deutschlands Flora. Nürnberg and Leipzig (Germany), 1798 - 1848.

E

Elench. fung. = Elenchus fungorum latine et germanice, etc. Halae (=Halle, Germany), 1783 - 1789.

En. pl. Saell. = Enumeratio plantarum in partibus Saellandiae septentrionalis et orientalis. Hafniae (=Copenhagen, Denmark), 1801 - 1803.

Engl. Jahrb. = Engler's Botanische Jahrbücher. Leipzig (Germany), 1881 - 1916 (?).

Epicr. = Epicrisis systematis mycologici, seu synopsis hymenomycetum. Upsaliae et Lundae (=Upsala and Lund, Sweden), 1836 - 1838.

F

Fl. B. et M. Congo = Études de Systématique et de Géographie Botaniques sur la Flore du Bas- et du Moyen-Congo. Brussels (Belgium), 1903 - 1912.

Fl. germ. = Testamen Florae germanicae. Lipsiae (=Leipzig, Germany), 1788 - 1802.

Flore Fr. = Flore Francaise. Paris (France), 1805 and 1815.

Formosa Bull. = Bulletin of the Division of Pathology of the Sugar Experiment Station. Daimokko (Formosa), 1911 - →

Fung. meck. sel. = Fungi mecklenburgenses selecti. Lünebergi (=Lüneberg, Germany), 1788 - 1802.

G

Grevillea = Grevillea. London (England), 1872 - 1894.

H

Handb. allg. Mykol. = Handbuch der allgemeinen Mykologie. Stuttgart (Germany), 1851.

Handb. Pflanzenkr. = Handbuch der Pflanzenkrankheiten. Berlin (Germany), 1886.

Hardw. Sc. Goss. = Hardwicke's Science Gossip for Students and Lovers of Nature. London (England), 1865 - 1902.

Hawaiian Pl. Rec. = Hawaiian Planters' Record. Honolulu (U. S. A.), 1909 - →

Hedwigia = Hedwigia. Dresden (Germany), 1852 - →

Herb. Fr. = Herbier de la France ou collection complette des plantes indigènes de ce royaume. Paris (France), 1780 - 1793.

Hist. Champ. Fr. = Histoire des Champignons de la France. Paris (France), 1791 - 1798.

Hook. Jour. Bot. = Hooker's Journal of Botany and Kew Garden Miscellany. London (England), 1849 - 1857.

H. S. P. A. = Bulletin of the Division of Pathology and Physiology, Experiment Station of the Hawaiian Sugar Planters' Association. Honolulu (U. S. A.), 1905 - → (Vols. 1 and 2 (Bulletins 1-12) issued under above title. Starting with Vol. 3, title changed to Bulletin of the Experiment Station, H. S. P. A., Botanical Series.)

I

Ic. fung. = Icones fungorum hucusque cognitorum. Abbildungen der Pilze und Schwämme. Pragae (=Präg, Austria, (now Czecho-Slovakia)), 1837 -1842.

Icon. descr. fung. = Icones et descriptiones fungorum quorumdam singularium. Ratisbonae (=Regensburg, Germany), 1761.

Infusionsth. = Die Infusionsthierchen. (Germany), 1838.

Isis = Sitzungsberichte der naturwissenschaftliche Gesellschaft Isis. Dresden (Germany), 1862 - →

J

Jahrb. nass. Naturk. = Jahrbucher des nassauischen Vereins für Naturkunde. Wiesbaden (Germany), 1863 - →

Jour. Bact. = Journal of Bacteriology. Baltimore (U. S. A.), 1916 - →

Jour. de Bot. = Journal de Botanique. Paris (France), 1808 - 1809.

Journal de Bot. = Journal de Botanique. Paris (France), 1887 - 1909.

Jour. Dep Agr. P. R. = Journal of the Department of Agriculture. San Juan (Porto Rico), 1917 - →

Jour. Linn. Soc. = Journal of the Linnaean Society of London, Botany. London (England), 1857 - →

Jour. Roy. Micros. Soc. = Journal of the Royal Microscopical Society of London. London (England), 1878 - →

K

K. Svenska Vet. Akad. Handl. = Kongliga Svenska Vetenskaps Akademiens Handlingar. Stockholm (Sweden), 1793 - →

Kew Bull. = Bulletin of Miscellaneous Information, Kew Gardens. London (England), 1887 - →

L

Linnaea = Linnaea, ein Journal für die Botanik in ihrem ganzen Umfange. Berlin (Germany), 1826 - →

M

Mag. Ges. nat. Freunde = Magazin der Gesellschaft der naturforschender Freunde zu Berlin. Berlin (Germany), 1801 - →

Malay Bull. = Bulletin of the Department of Agriculture of the Federated Malay States. Kuala Lumpur (F. M. S.), 19— - →

Malpighia = Malpighia. Genoa and Catania (Italy), 1887 - 1913.

Mededeel. Plant. = Mededeelingen uit s'Lands Plantentuin. Batavia (Java), 1884 - →

Mededeel. Suiker. = Mededeelingen van het Proefstation voor Suikerreit in West-Java. Kagok-Tegal (Java), 1890 - 1892.

Mem. Dep. Agr. India = Memoirs of the Department of Agriculture in India, Botany. Calcutta (India), 1906 - →

Michelia = Michelia. Patavii (=Passau, Germany), 1877 - 1881.

Microorg. = Die Microorganismen. Leipzig (Germany), 1896.

Monsunia = Monsunia. Leipzig (Germany), 1899 - ?

Mycologia = Mycologia. New York (U. S. A.), 1909 - →

N

N. Am. Fl. = North American Flora. New York (U. S. A.), 1905 - →

Nuovo Giorn. bot. Ital. = Nuovo Giornale botanico Italiano. Firenze (= Florence, Italy), 1869—?

O

Outl. = Outlines of British Fungology. London (England), 1860.

P

Par. Alg. Pil. Javas = Parasitische Algen und Pilze Javas. Batavia (Java), 1900.

Prachtflora = Prachtflora europäischer Schimmelbildungen. Leipzig and Dresden (Germany), 1839.

Prae. Monogr. Hend. = Praecursor ad Monographiam Hendersoniae. In Vol. X of Nuovo Giorn. bot. Ital., q. v.

Prim. Fl. esseq. = Primitiae Florae essequeboensis. Goettingae (=Göttingen, Germany), 1828.

R

Rech. Veg. Inf. = Recherches pour Servir a l'Histoire Naturelle des Végétaux Inférieurs. Paris (France), 1874 - 1886.

Rep. Dep. Agr. N. S. W. = Report of the Department of Agriculture. Sydney (New South Wales), 1893 - →

Rev. Agr. Vet. = Revista de la Facultad de Agronomia y Veterinaria. La Plata (Argentina), 1905 - →

Rev. Mus. La Plata = Revista del Museo de La Plata. La Plata (Argentina), 1890 - →

S

Sluzow. Monogr. = Sluzowce (Mycetozoa) Monografia. Paris (France), 1875 - 1876.

Sp. plant. = Species plantarum exhibentes plantas rite cognitas, etc. Holmiae (=Stockholm, Sweden), Vienna (Austria) and Berlin (Germany), 1753 - 1838.

Summa veg. Scand. = Summa vegetabilium Scandinaviae, etc. Holmiae (=Stockholm, Sweden) and Lipsiae (=Leipzig, Germany), 1846 - 1849.

Syll. cryptog. = Sylloge generum specierumque cryptogamarum, quas in variis operibus descriptas, etc. Paris (France), 1856.

Syll. fung. = Sylloge fungorum omnium hucusque cognitorum. Patavii (=Passau, Germany), Berolini (=Berlin, Germany) and Parisiis (=Paris, France), 1882 - 1913.

Sylv. myc. berol. = Sylvae mycologicae berolinenses. Berolini (=Berlin, Germany), 1818.

Symb. mycol. = Symbolae mycologicae, in Jahrb. nass. Naturk., q. v.

Syn. meth. fung. = Synopsis methodica fungorum, sistens enumerationem omnium hucusque detectarum specierum, etc. Goettingae (=Göttingen, Germany), 1801.

Syst. myc = Systema mycologicum, sistens fungorum ordines, genera et species, etc. Gryphiswaldiae (=Greifswald, Germany), 1821 - 1832.

Syst. nat. = Systema naturae, sive regna tria naturae systematica proposita, etc. Lugduni Batavorum (=Leiden, Holland), Holmiae (=Stockholm, Sweden), Halae and Lipsiae (=Halle and Leipzig, Germany), 1735 - 1791.

Syst. P. u. S. = Das System der Pilze and Schwämme. Würzburg (Germany), 1817.

Syst. Pilze = Das System der Pilze. Bonn (Germany), 1837 - 1858.

T

Tr. Am. Phil. Soc. = Transactions of the American Philosophical Society. Philadelphia (U. S. A.), 1771 - →

Trans. Linn. Soc. Lond. = Transactions of the Linnaean Society of London, Botany. London (England), 1791 - →

Trans. Linn. Soc. N. S. W. = Transactions of the Linnaean Society of New South Wales. Sydney (N. S. W.), 1875 - →

U

Unters. Ges. Mykol. = Untersuchungen aus dem Gesammtgebiet der Mykologie. (Hefte 1-5 are entitled Botanische Untersuchungen über Schimmelpilze.) Leipzig and Münster (Germany), 1872 - 1912.

Z

Ziekt. Suik. = De Ziekten van het Suikerreit op Java. Leiden (Holland), 1898.

Note—The symbol → indicates that the publication is still in progress.

EXPLANATION OF PLATE 1.

MOSAIC IN LAHAINA CANE.

Figure 1—Portion of leaf showing the characteristic mottling induced by Mosaic. ½ natural size.

Figure 2—The corresponding portion of a healthy leaf. ½ natural size.

EXPLANATION OF PLATE 2.

MOSAIC IN LAHAINA CANE.

Corresponding portions of two sticks from adjacent stools. The cuttings from which these stools grew were planted at the same time and the sticks, when cut, had attained about equal development. Figures ½ natural size.

Figure 1—Portion of a stick showing the mottling of the rind induced by Mosaic. Diseased shoots of this variety do not always show mottling of the rind to the extent exhibited by this specimen.

Figure 2—The corresponding portion of a healthy stick.

PLATE [illegible]

1

2

EXPLANATION OF PLATE 3.

Mosaic and Sereh.

Figure 1—Yellow Caledonia, 7/9 natural size. The irregular grooving of the rind as seen on the two lower internodes and the discoloration of the internal tissues as seen in the upper split portion are symptoms induced in the canes of this variety by Mosaic.

Figure 2—A Java seedling, G. Z. 247 B., ½ natural size. Portion of a serehed stick split lengthwise to show the conspicuous red streaks which are vascular bundles filled with a red gum. In Java such red, gum-filled bundles are considered the critical symptom of Sereh. The development of adventitious roots on the aerial portion of the stick is also a characteristic symptom of Sereh in this cane variety.

PLATE 3

EXPLANATION OF PLATE 4.

Corn Mosaic:

Figure 1—A portion of diseased leaf of the sweet corn variety, Stowell's Evergreen. An advanced stage of the disease. The leaf is lighter in color than are normal leaves of the same age.

Figure 2—A portion of a leaf of the field corn variety, Boone County White. An early stage of the disease. The light green stripes on the normal green background are distributed at regular intervals over the portion of the leaf bordering on the mid-rib; the edges are still healthy.

Figure 3—A portion of a diseased leaf of the sweet corn variety, Early Fordhook. The light green stripes are shorter than those shown in figures 1 and 2.

Figure 4—A portion of a diseased leaf of the field corn variety, U. S. Selection No. 120. This shows the typical mosaic pattern. The line of demarkation between light and dark green areas not so sharp as is shown in figures 1, 2, and 3.

EXPLANATION OF PLATE 5.

The Intracellular Bodies of Corn Mosaic:

Figure A—An intracellular body closely applied to a host cell nucleus. The large vacuole and the pointed side lobe are characteristic of one stage of the disease.

Figure B—A body attached to its host cell nucleus by means of a thin, veil-like structure. The chromatin of the enlarged nucleus stains deeply and occurs as globular masses around the nucleolus.

Figure C—A foreign body in a typical position on a host cell nucleus. It fits over the nucleus like a cap.

Figure D—A body containing deep staining granules but no vacuoles.

Figure E—An early stage in the development of the body. It is irregular in shape and shows a coarse structure.

Figure F—A large foreign body closely applied to its host cell nucleus. In it are two large vacuoles and a number of deep staining granules.

Figure G—A structure peculiar to one stage of the disease. This structure distinguishes the bodies from all other cell inclusions.

Figure H—A body containing many small vacuoles.

Figure I—A picture drawn from living material. The cell contains two nuclei; one is shown with a body closely attached; the other is hidden from view by the rather large body at the right. The rapid streaming of the cytoplasm in such cells is a marked symptom of the disease. $\times$ 365.

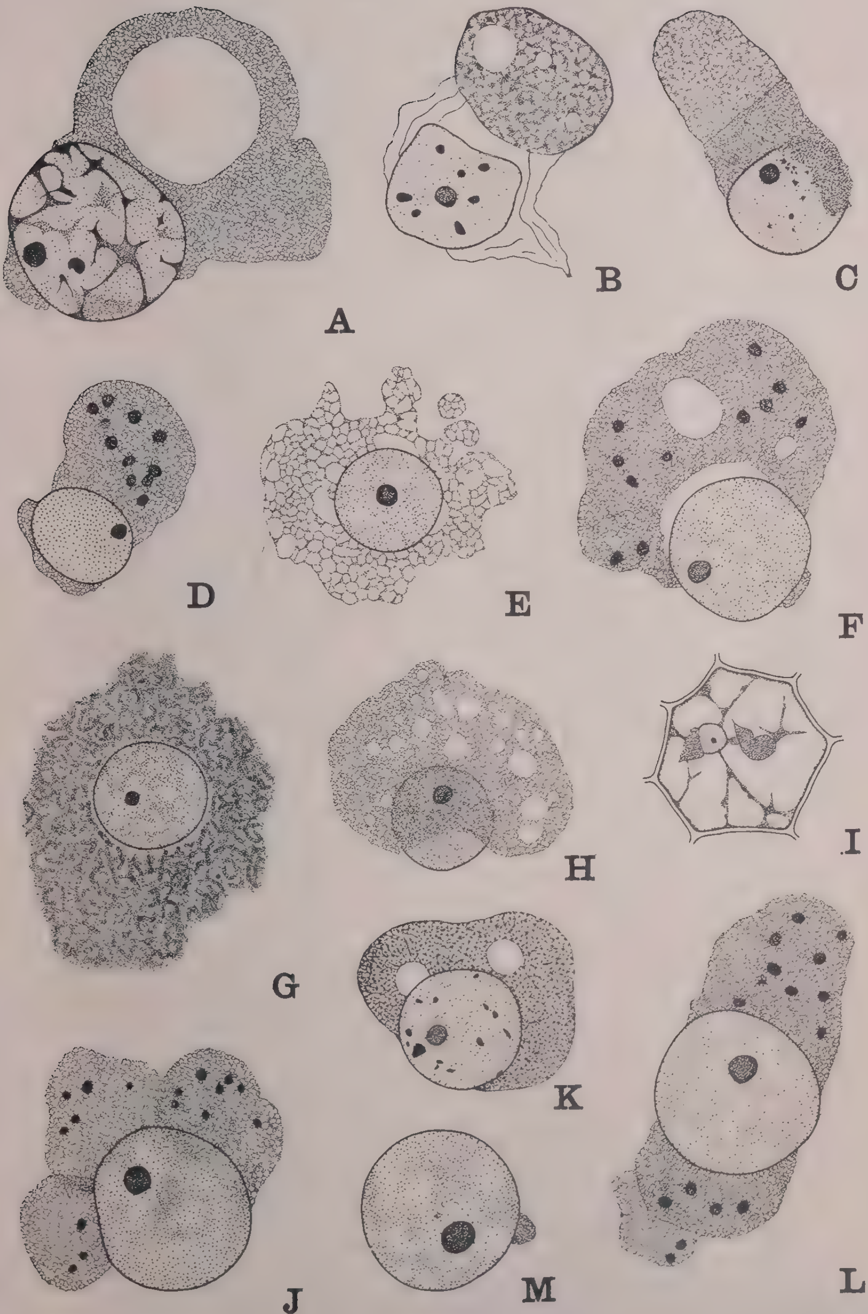
Figure J—A much enlarged host cell nucleus with four of the bodies clustered around it. The bodies contain deep staining granules.

Figure K—A body containing two vacuoles and showing dense structure. A typical position on the host cell nucleus.

Figure L—An elongated body in another typical position on a host cell nucleus. This body resembles the one shown in figure C.

Figure M—A tiny foreign body attached to an enlarged host cell nucleus. The cell containing this body showed an early stage of the disease. All $\times$ 1825 except figure I.

PLATE 5.



EXPLANATION OF PLATE 6.

Corn Mosaic on the Variety Boone County White:

Figures A and B—Short pieces of healthy and diseased stalks. The dark areas shown on the cut ends of diseased stalks represent sections through necrotic tissue and elongated cavities. The collapse of such diseased tissues causes shrivelling of the stalk.

Figure C—A portion of a diseased leaf, showing striping along the veins.

Figure D—A mosaic plant. The height of the plant may be judged by the two-foot ruler shown in the picture. The shortening of the upper internodes has caused a bunching of the leaves at the top of the plant.

Figure E—A healthy plant of the same age as the diseased plant shown in figure D.


A
B
C
D
E

EXPLANATION OF PLATE 7.

Corn Mosaic:

Figure A—A diseased and healthy plant of the popcorn variety, White Rice. The two-foot ruler shows the heights of the plants. The diseased plant is much dwarfed; its stalk is shrivelled and distorted.

Figure B—A portion of a diseased leaf of the field corn variety, Reid's Yellow Dent. Typical striping is shown along some of the veins of this leaf.

Figure C—A diseased and healthy plant of Reid's Yellow Dent. The diseased plant is much dwarfed and its leaves are bunched at the top.

PLATE 7.

EXPLANATION OF PLATE 8.

Corn Mosaic:

A diseased and healthy plant of the sweet corn variety, Early Fordhook. Both plants were cut at the surface of the soil; a two-foot ruler shows their heights. The leaves have been removed in order to show the shortening of the internodes of the diseased plant. The two plants grew in the same row; the healthy one has produced a fair sized roasting ear, while the diseased plant has failed to give even a small nubbin.

PLATE 8.

EXPLANATION OF PLATE 9.

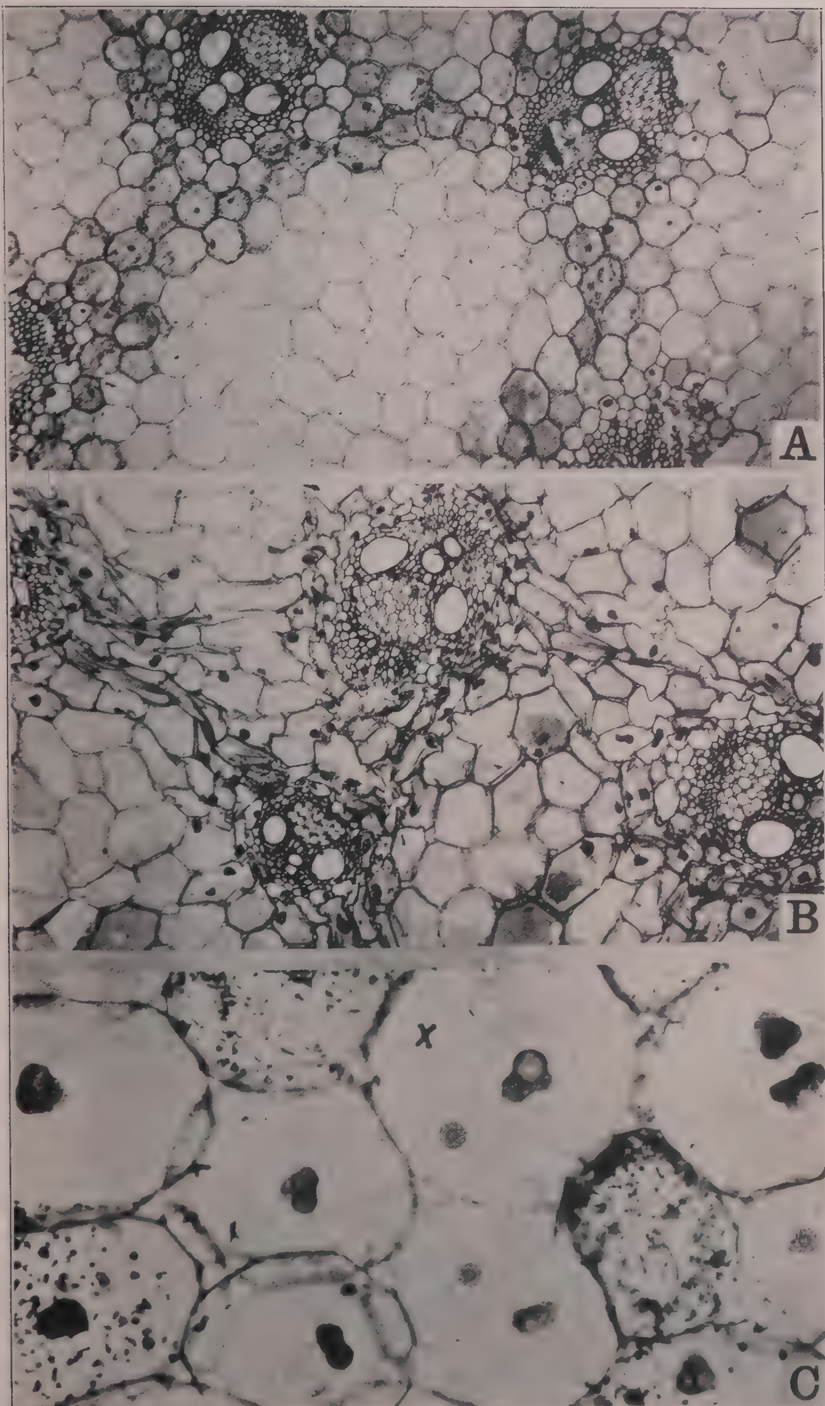
Corn Mosaic:

Figure A—A portion of a cross section of diseased stalk tissue. The relation between diseased and healthy tissue is clearly shown. The walls of cells that contain the foreign bodies are slightly thickened and are beginning to break down. Cells that are free from the bodies appear healthy. The disease seems to have spread from the fibro-vascular bundles. × 80.

Figure B—A cross section of stem tissue similar to that shown in A. Cells that contain the bodies show various stages of collapse; cells that do not contain them are healthy. × 80.

Figure C—A section of stem tissue highly magnified. The body in the cell marked X is the same as that shown in Plate 5, figure A. Although the bodies in these cells are quite large, the cells have not yet started to enlarge or to break down. × 320.

PLATE 9.



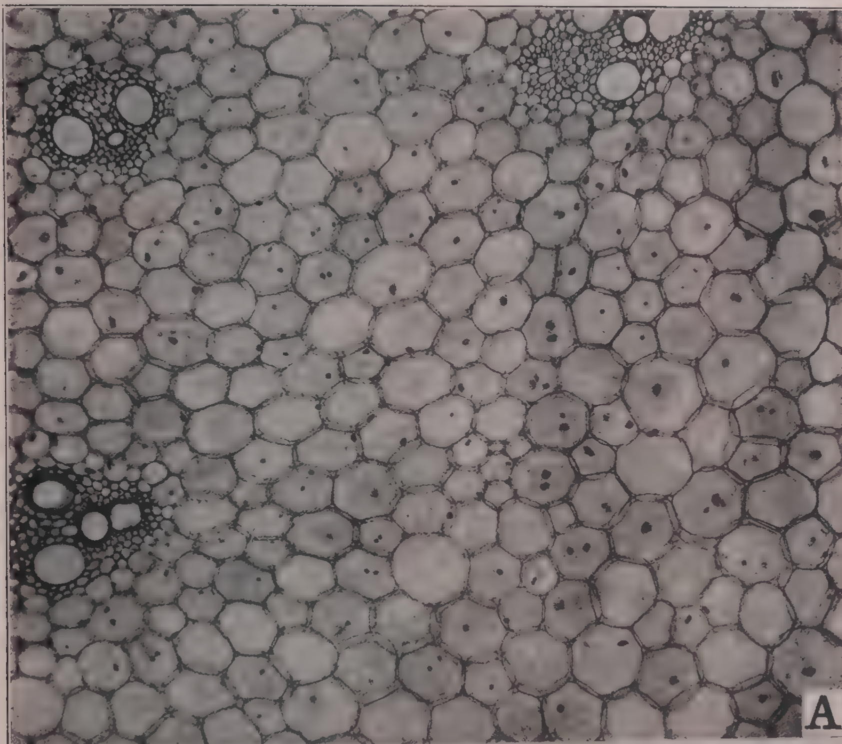
EXPLANATION OF PLATE 10.

CORN MOSAIC:

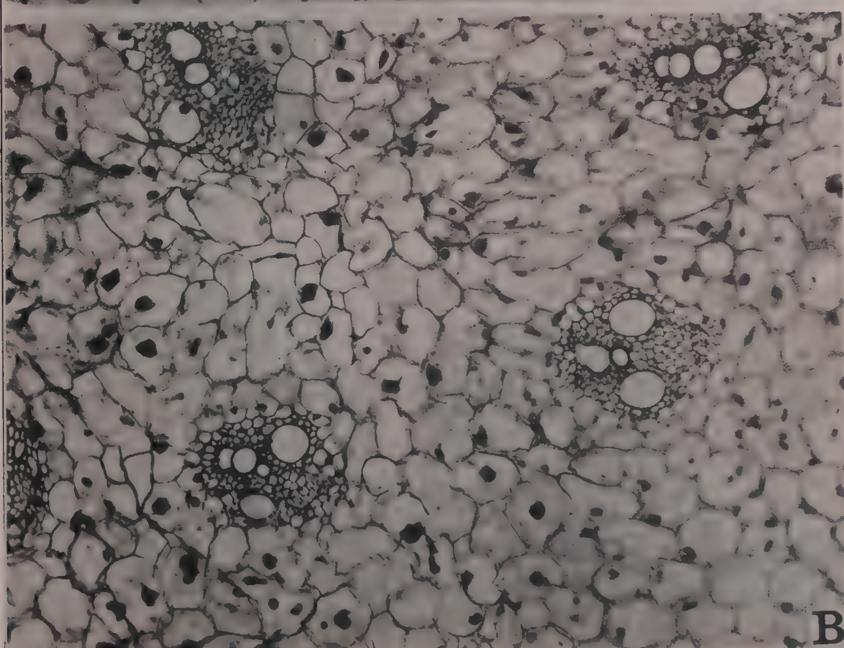
Figure A—A cross section of stem tissue through which the disease has spread. The foreign bodies shown here are small and the tissues are only slightly diseased. $\times$ 80.

Figure B—A cross section of stem tissue similar to that shown in figure A, but the bodies are larger. Many of the cells are growing and the tissues show early stages of necrosis. $\times$ 80.

PLATE 10.



A



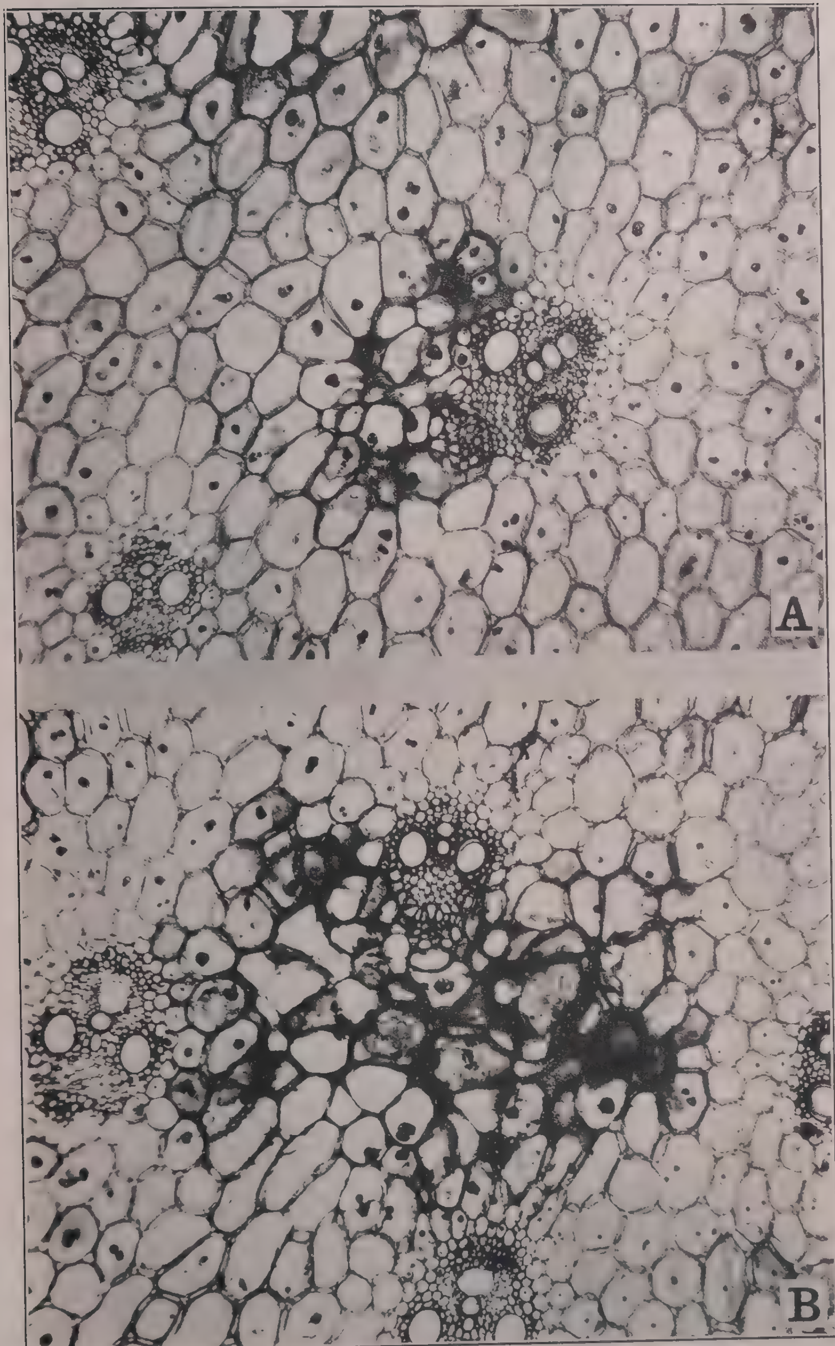
B

EXPLANATION OF PLATE 11.

CORN MOSAIC:

Figure A—A cross section of diseased stalk tissue, showing an early stage of necrosis near a fibro-vascular bundle. The cells bordering on the necrotic tissues are slightly enlarged and contain foreign bodies that are larger than those in the cells of the surrounding tissues. $\times 80$.

Figure B—A cross section of stalk tissue showing a somewhat more advanced stage in the breaking down of diseased cells. The walls appear to be softened; many cells have collapsed but no actual cavity has yet been produced. The bodies in the cells that are breaking down are larger than those in the cells of surrounding tissues. $\times 80$.


A
B

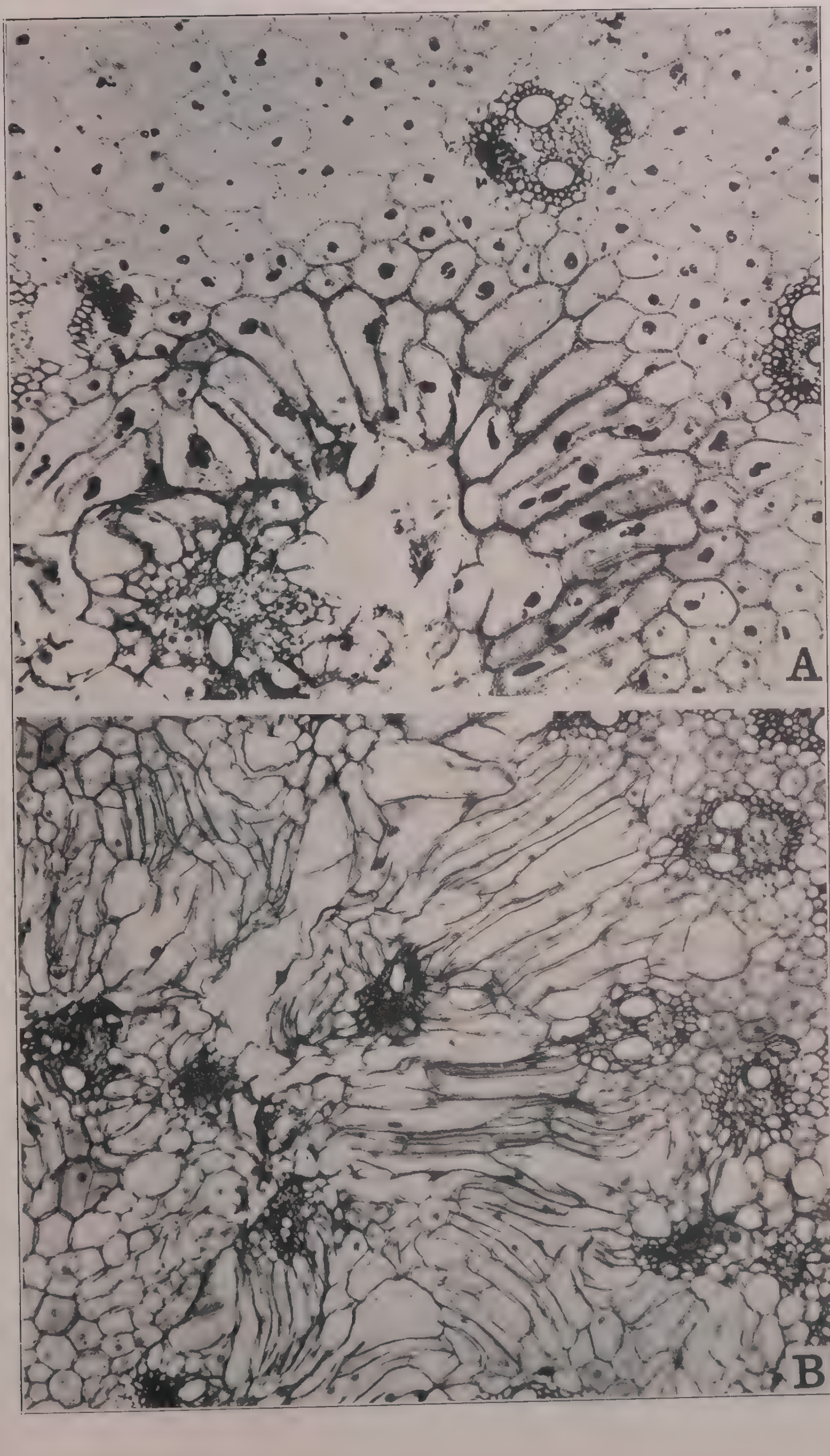
EXPLANATION OF PLATE 12.

CORN MOSAIC:

Figure A—A cross section of stalk tissue showing an early stage in the formation of an internal cavity. The cells bordering the cavity are much enlarged and their walls appear softened and thickened. Here again the bodies in the cells bordering on the cavity are larger than those in the cells of the surrounding tissues. $\times$ 80.

Figure B—A section showing an early stage of necrosis in young stem tissue. Although the foreign bodies are small and inconspicuous, many of the cells have responded to the growth stimulus. $\times$ 80.

PLATE 12.



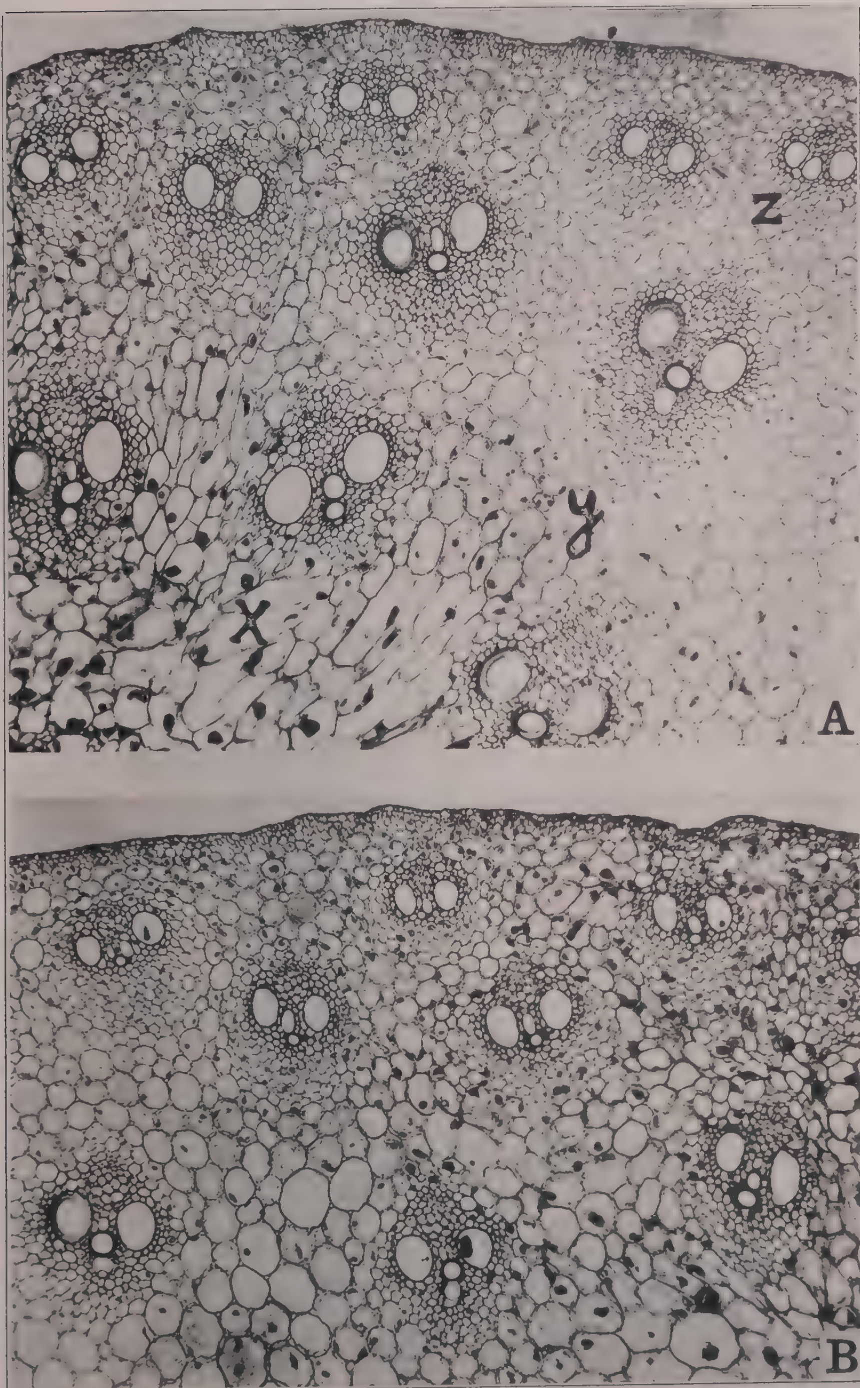
EXPLANATION OF PLATE 13.

Corn Mosaic:

Figure A—A cross section of stalk tissue in which the disease seems to be spreading. In the region marked X the bodies are large. Here the host cells are making abnormal growth. Many cells are being crushed by the pressure thus produced. In the region marked Y the intracellular bodies are small and the host cells are little affected by the disease. The cells in the region marked Z do not contain the bodies. These cells stain normally and appear to be healthy. $\times$ 80.

Figure B—A cross section of stalk tissue similar to that in figure A, showing progressive stages of the disease. $\times$ 80.

PLATE 13.



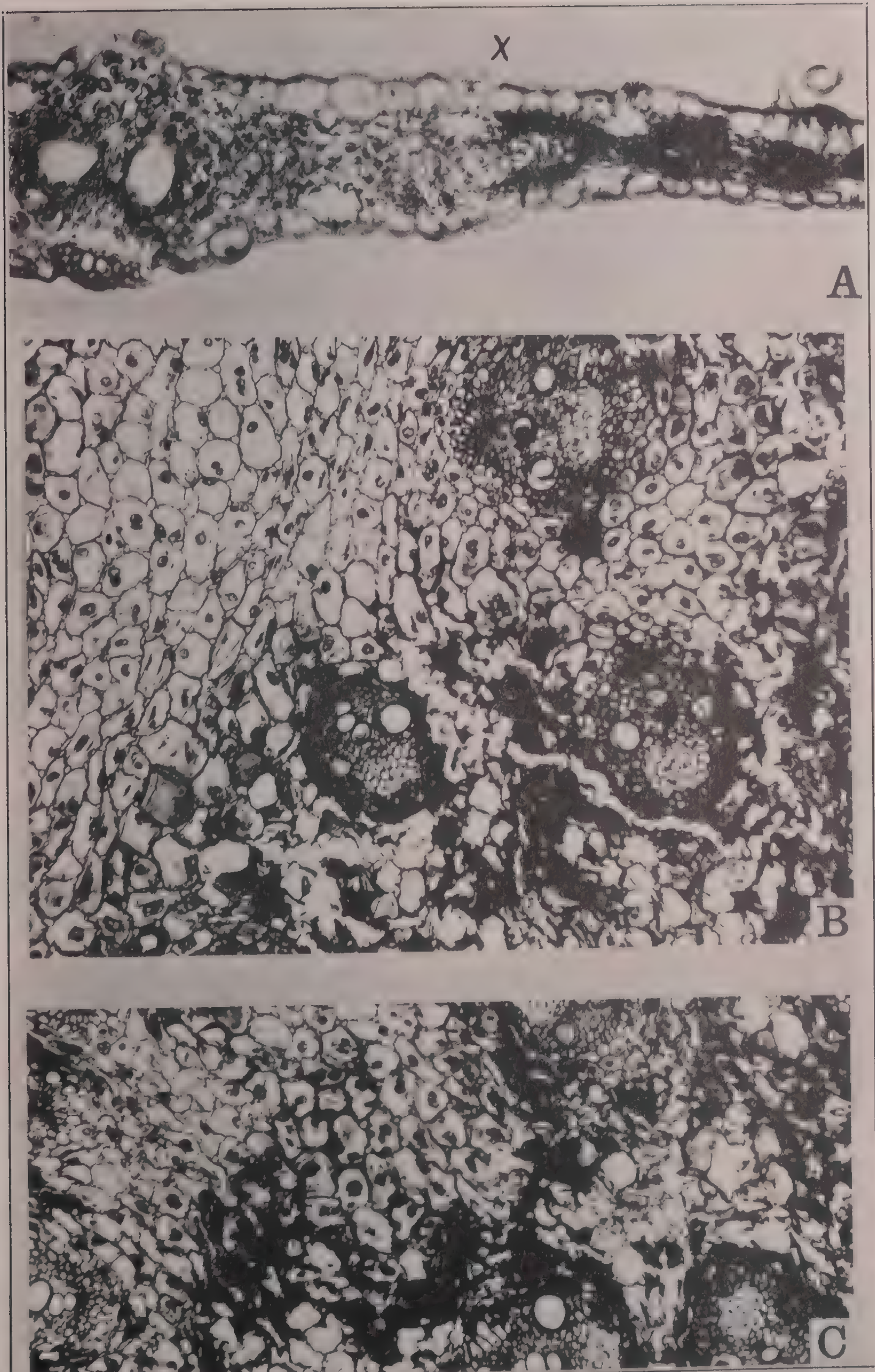
EXPLANATION OF PLATE 14.

Corn Mosaic:

Figure A—A cross section of leaf tissue. That portion of the leaf on the right of the letter X was dark green in color and is healthy; the portion on the left was light green in color and is diseased. The cells in the diseased portion contain the foreign bodies, which, however, are not clearly shown in the picture. × 150.

Figure B—A section through stem tissue showing a late stage of disease. × 80.

Figure C—A section of stalk tissue similar to that shown in B. At this stage of the disease, most of the cells of the parenchymatous tissues have collapsed. × 80.


X
A
B
C

EXPLANATION OF PLATE 15.

Corn Mosaic:

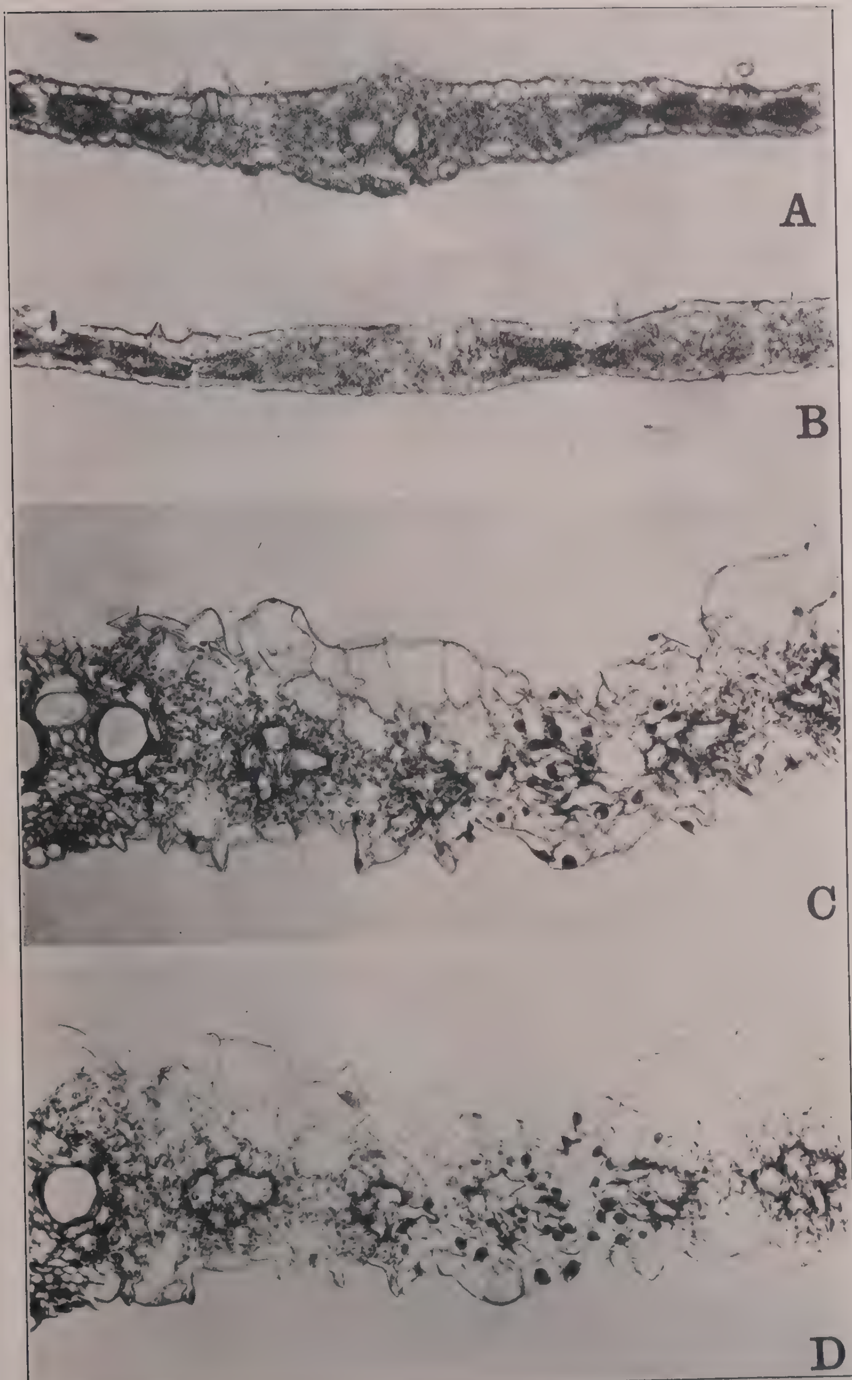
Figure A—A cross section of diseased leaf tissue. The light colored tissues bordering on the large fibro-vascular bundle are diseased; the dark colored tissues on either side are healthy. Foreign bodies are present in the diseased cells. $\times$ 60.

Figure B—A cross section of diseased leaf tissue. The parts that appear dark in the photograph are healthy; the lighter colored portions are diseased. The cells in the diseased portions have started to grow, causing the leaf to thicken in places. $\times$ 60.

Figure C—A cross section of diseased leaf tissue highly magnified. The foreign bodies appear as dark objects in the photograph. They are present in the diseased cells but absent from the cells of the healthy tissues. $\times$ 150.

Figure D—A cross section of diseased leaf tissue similar to that shown in figure C. Here again the cells in the tissues that were light green in color contain the foreign bodies while the cells in the healthy deep green colored tissues on either side are free from them. $\times$ 150.

PLATE 15.



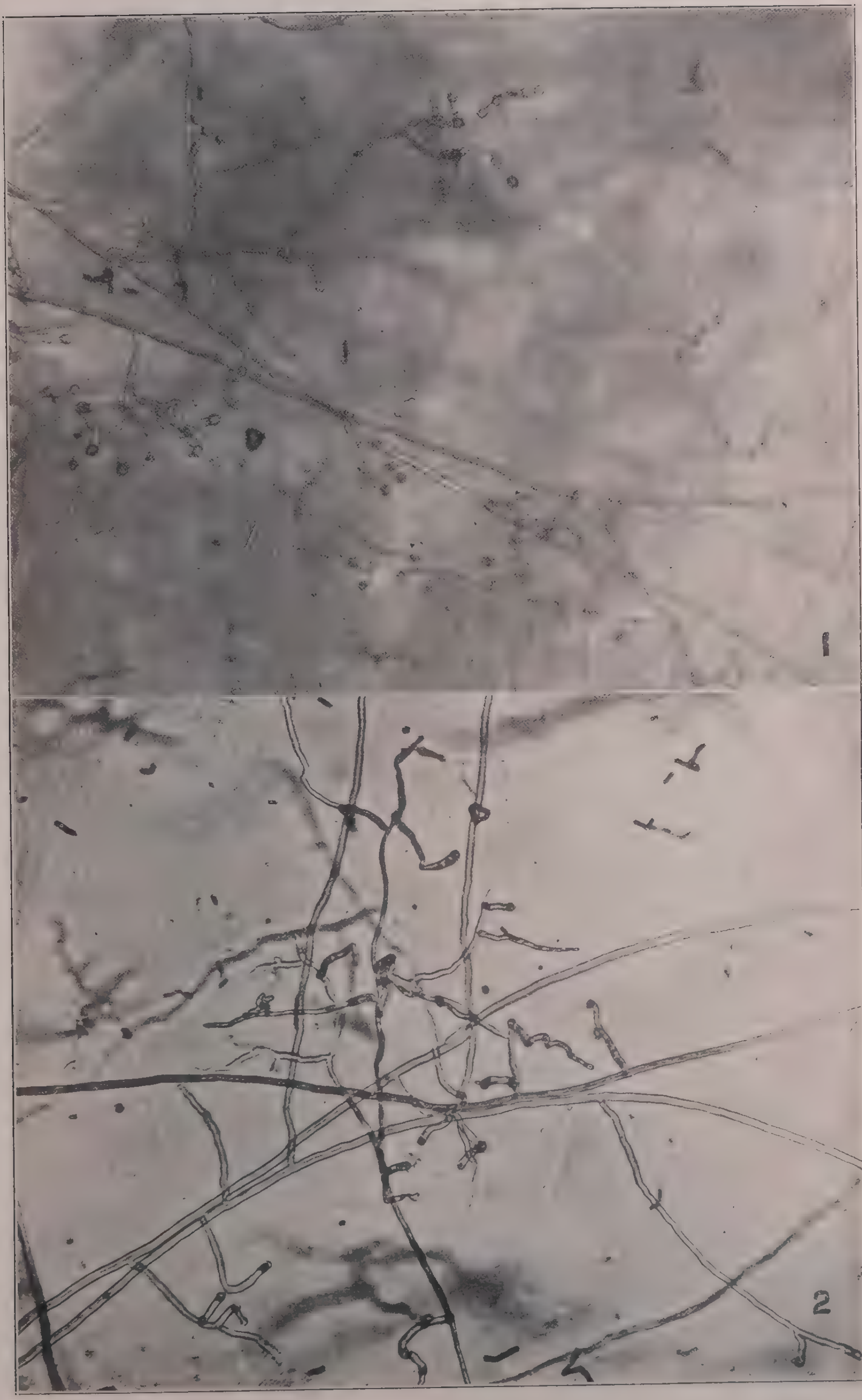
EXPLANATION OF PLATE 16.

Characteristic habit growth of young hyphae of *Pythium Butleri* from cane and pineapples. Note the short knobbed branches which suggest spores. The tips are not walled off, however. $\times$ 200.

Figure 1—*P. Butleri* from cane, isolated from natural root infection of Lahaina cane plant in sick Waipio soil, pot culture.

Figure 2—*P. Butleri* from pineapple. Isolated from collapsed roots of a plant from Kailua, Oahu.

PLATE 16.



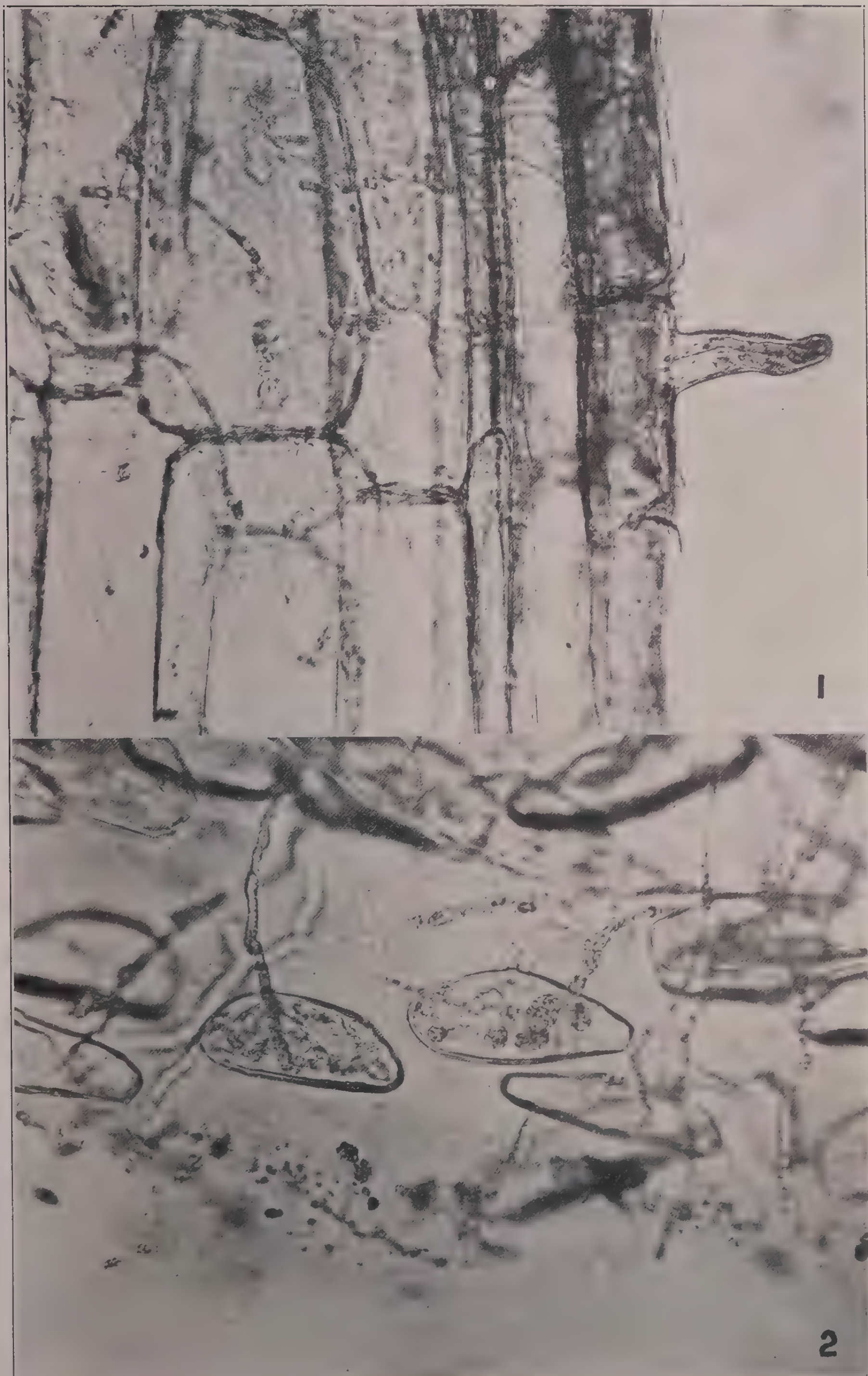
EXPLANATION OF PLATE 17.

Pythium Butleri in root cells.

Figure 1—Section of Lahaina cane root from Waipio. Root cells and young root-hair showing mycelium of the *Pythium* type. $\times$ 500.

Figure 2—Cells of root-cap occupied by the same sort of mycelium. $\times$ 500.

PLATE 17.



EXPLANATION OF PLATE 18.

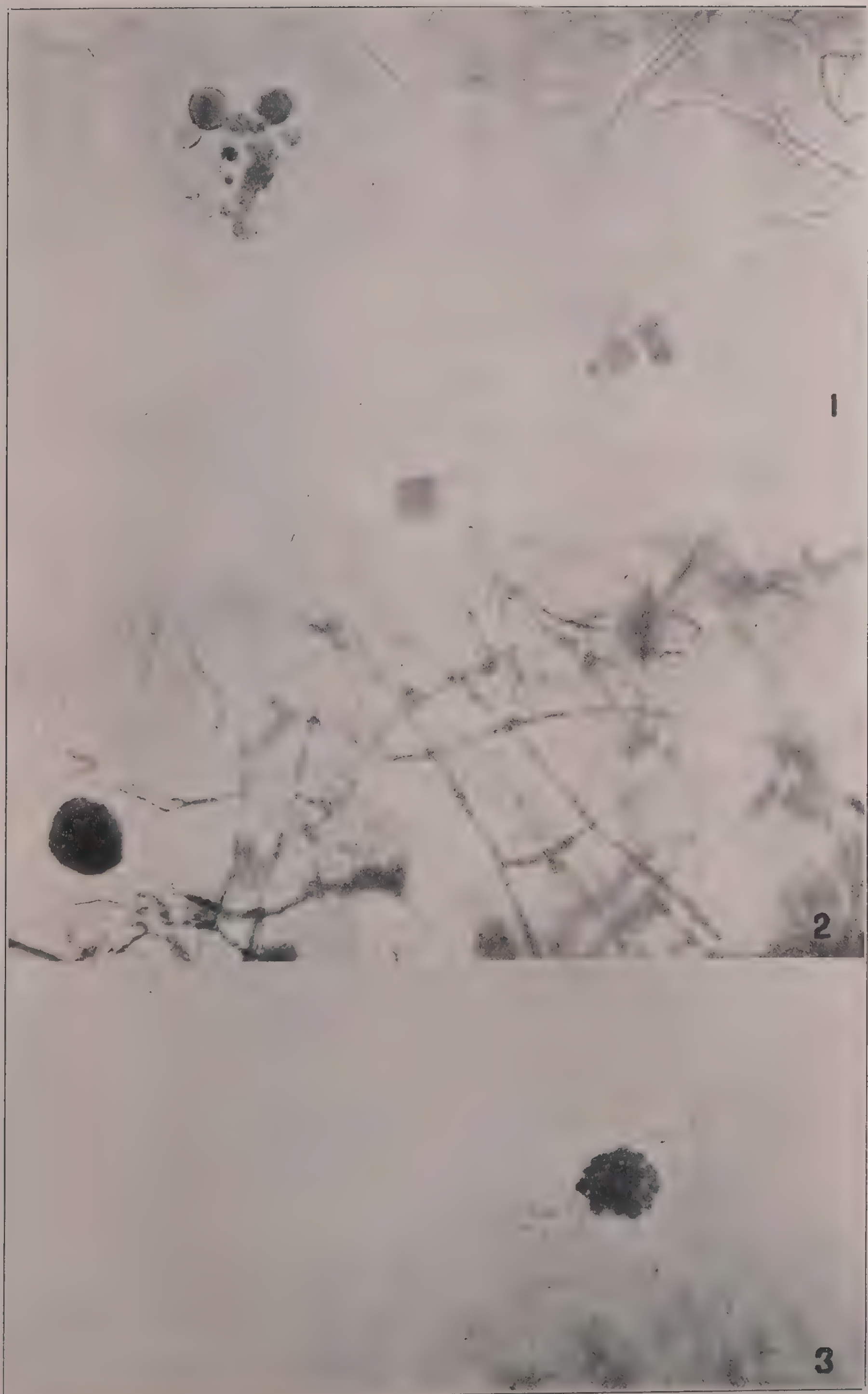
Features of life cycle of *Pythium Butleri* from cane. Pure culture studies.

Figure 1—Mycelium and presporangia, water culture from oat agar plates. $\times$ 200.

Figure 2—The emptied presporangium and the dark sporangia; the darker sporangium has just completed its streaming from the presporangium. Water culture, cane root. $\times$ 200.

Figure 3—Zoospores in hyalin zoosporangium inactivated with osmic acid. Water culture, cane root. $\times$ 200.

PLATE 18.



EXPLANATION OF PLATE 19.

Pure culture life cycle studies of *Pythium Butleri* from cane (continued).

Figure 1—Maturing zoosporangium with its hyalin wall, more highly magnified. Cleavage lines becoming evident. × 500.

Figure 2—Oospores developing. Note the several antheridia about one oogonium. A few days old oat agar culture. × 500.

PLATE 19.

EXPLANATION OF PLATE 20.

Water culture of *Pythium Butleri* on Lahaina cane root. $\times$ 500.

Figure 1—Optical section of young root showing intracellular swollen protoplasmic bodies which are thought to be presporangia.

Figure 2—Hyphae passing from presporangium-like bodies through cell wall to surface of root.

Figure 3—Empty presporangium on surface of root.


1
2
3

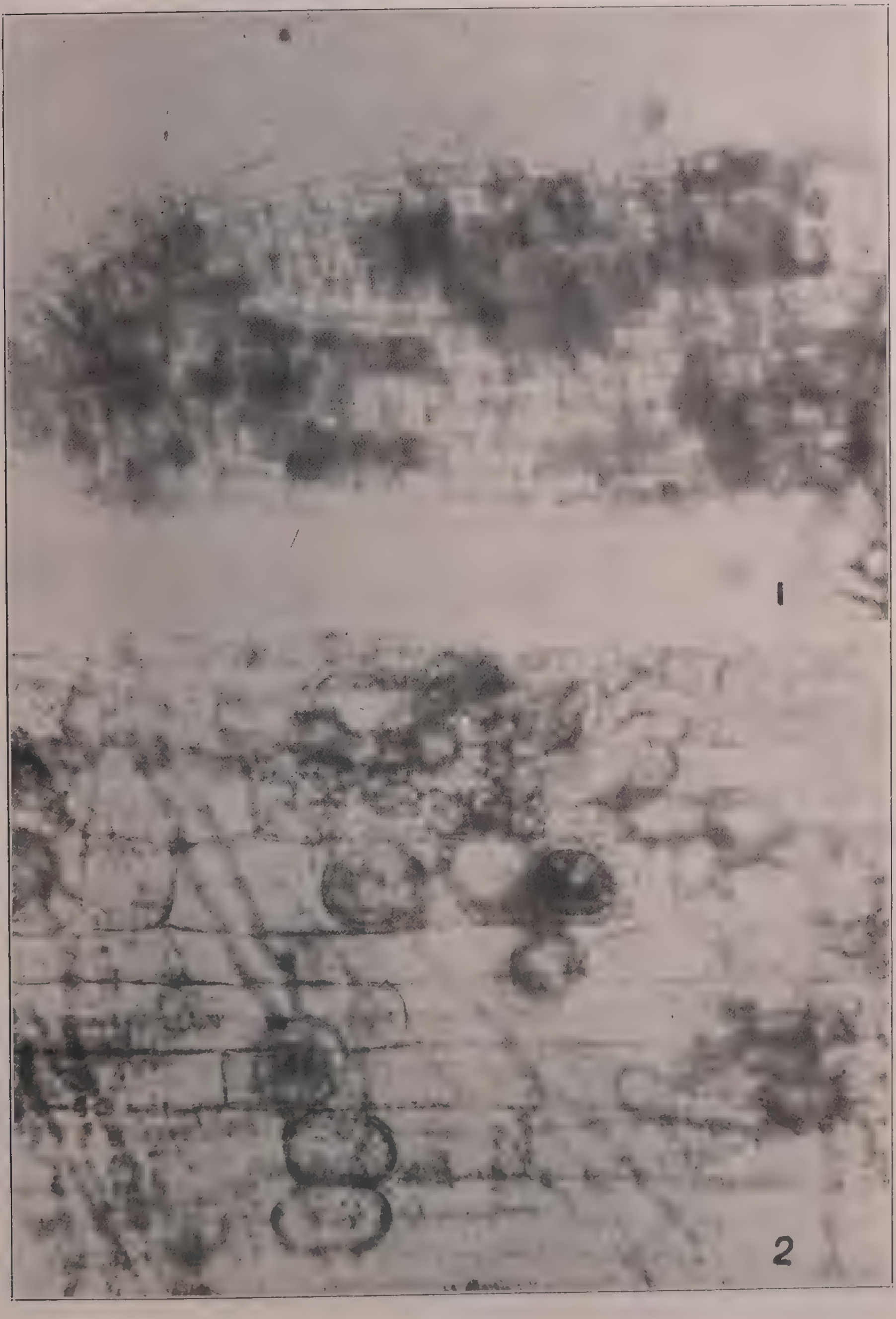
EXPLANATION OF PLATE 21.

Optical section of young cane roots from water cultures of *Pythium Butleri.*

Figure 1—Shadowgraph of infected young root. The dark areas represent the swollen presporangium-like mycelium and oospore content of the cells. × 200.

Figure 2—Closer view of same sort of material. Oospores in the center and presporangium-like swollen mycelium above. × 500.

PLATE 21.



EXPLANATION OF PLATE 22.

The asexual life cycle of a pure culture of *Pythium Butleri* from cane roots, drawn from camera lucida sketches.

Figures 1–4—Presporangia formed at end of hyphae.

Figure 1—Four-day water culture. × 350.

Figures 2–3—Four-day water culture. × 500.

Figure 4—Oat agar culture 13 days and water culture 2 days. × 500.

Figures 5–12—Development of sporangium from the presporangium and maturing of the former. Drawing from sketches made of same sporangium at times indicated. × 500.

Figure 5—February 1, 1921, 11:10 A. M. Streaming of presporangium into vesicle completed. Empty presporangium below.

Figure 6—11:13 A. M.

Figure 7—11:16 A. M.

Figure 8—11:20 A. M. Cleavage beginning.

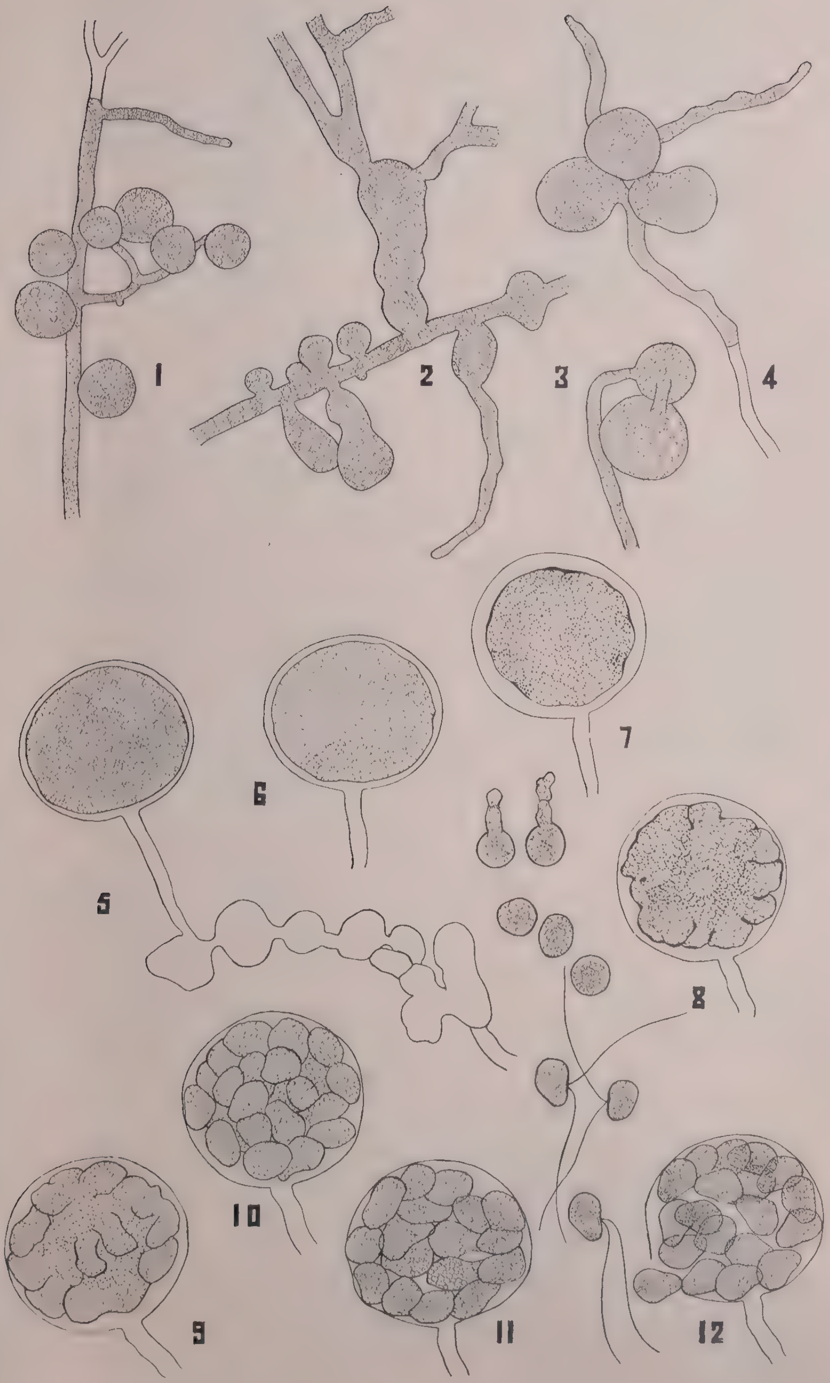
Figure 9—11:23 A. M. Rocking movement of entire content beginning.

Figure 10—11:24 A. M. Rocking of individual zoospores beginning. Differentiation completed.

Figure 11—11:27 A. M. Active, oscillatory individual movement of zoospores present. They ruptured the vesicle and escaped at 11:27 A. M.

Figure 12—The escape of the biciliate zoospores. Above are sketched the zoospores rounding up and germinating after the motile period.

PLATE 22.



EXPLANATION OF PLATE 23.

Morphology of *Pythium spp.*

Figures 1–6—*P. Butleri* from cane. Development of oospores.

Figure 1—Oogonium and antheridia. × 500. From string bean agar culture. Six hours later the oospore was distinctly walled, the appearance being similar to that of figure 5.

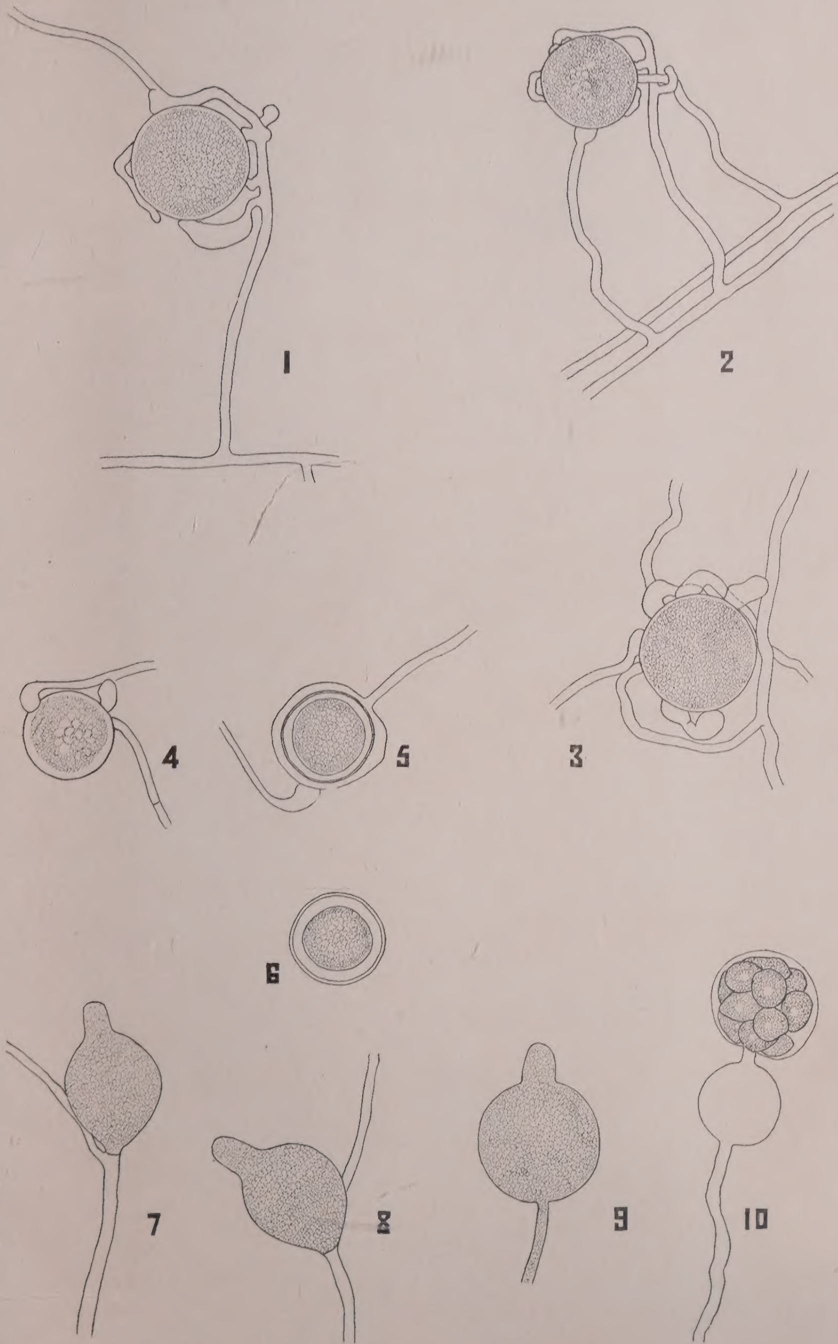
Figure 6—Oospore freed from oogonium.

Figures 7–10—*Pythium* sp., the cause of rot of taro. Asexual stage. × 500.

Figures 7–9—Sporangia formed at tips of sporangiophores, which continue growth, pushing spore to one side.

Figure 10—The contents of a sporangium (presporangium) have flowed out through the beak into the vesicle in which the zoospores are differentiated (the true sporangium).

PLATE 23.



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